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CLINICAL EXPERIENCE WITH A NEW ANTICOAGULANT, SINTROM (G 23350)

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As indicated by numerous publications[1,2] over the last 16 years, the use of coumarin drugs in anticoagulant therapy has been thoroughly investigated. However, the search for a so-called 'ideal' anticoagulant drug has continued. Such a 'prothrombin' depressant should meet the following requirements:[3]

1. It should act rapidly in lowering the prothrombin index to a therapeutic level.
2. Single daily doses should suffice to prevent fluctuations of the prothrombin index.
3. It should be metabolized or excreted quickly enough to allow a rapid return to normal of the prothrombin index after cessation of therapy.
4. A suitable pharmacological antagonist should rapidly counteract its effect in case of emergency.
5. The doses should be relatively constant in a given patient and from patient to patient.
6. Oral administration should be effective.
7. It must be non-toxic and well tolerated in therapeutic dosages.
8. It should be satisfactory for use as an ambulatory drug.

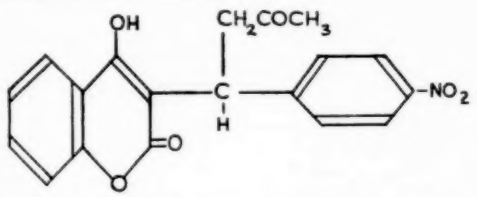
In addition, the cost of the drug should not be prohibitive.

At present, no drug available meets with all these requirements, but a new coumarin derivative, nitro-phenyl-acetyl-ethyl-4-hydroxycoumarin, commercially known as Sintrom* (G 23350), which was synthesized by Stoll and Litvan,[4] is stated to approach the 'ideal' prothrombin depressant more closely than many of the other anticoagulant drugs.[3]

Sintrom has to date not been used in South Africa and the purpose of this paper is to report our experiences with the use of this drug.

Chemical Properties (as determined by Montigel and Pulver[6])

Sintrom is a 3-(α-[4'-nitrophenyl]-β-acetyl-ethyl)-4-hydroxy-coumarin. The chemical formula is $C_{19}H_{15}NO_6$. It has the following structural formula:



It is a crystalline light-brown powder, which is almost tasteless and odourless. The molecular weight is 353 and the melting point 191-192°C. It is slightly soluble in organic solvents and in water, and becomes increasingly soluble in water as the pH is raised. It is a weak acid and is chemically stable. With alkalis it forms water-soluble salts.

Mode of Action

The mode of action and metabolism of sintrom have been thoroughly investigated by European[5,6] and American[3,7,8] workers. As with other coumarin derivatives, it causes a depression of factor VII (stable factor or pro-convertin) activity and to a lesser extent of prothrombin, early in the course of the treatment.[9] Both are equally affected after several weeks of treatment.[10] Factor V (labile factor or pro-accelerin) and antihaemophilic globulin are not influenced.[6,10] It probably also acts as an antagonist of vitamin K.[8] Sintrom, like other coumarins, is active only *in vivo* and not *in vitro*.[17]

Sintrom rapidly disappears from the body. It is excreted by the kidneys in an unaltered state, in contradistinction to other coumarins such as tromexan, which is converted to an inactive tromexan acid.[6] Degradation products of sintrom are found only in small quantities.[6]

To investigate the distribution of sintrom and dicoumarol in the body, Montigel and Pulver[6] administered 100 mg./kg. of sintrom or 20 mg./kg. of dicoumarol orally to rabbits each day, over a period of 4-6 days. The rabbits were killed 8 hours after the last dose and the amount of drug in each organ estimated. They found that dicoumarol accumulated not only in the blood but also in the tissues; the concentration in the liver was, on an average, 1/3rd of that in the blood. In contrast, sintrom was found in larger amounts in the liver than in the blood. The other organs, e.g. brain, heart, spleen, muscle and fatty tissues, contained only relatively small amounts of dicoumarol (0·5 - 2·3 mg.%) and sintrom (0·2 - 1·2 mg.%)

Montigel and Pulver found that if they injected rabbits intravenously with sintrom, tromexan or dicoumarol using doses of 5 mg./kg., the blood concentration fell from 3·5 mg.% to 0·2 mg.% in 2 hours with tromexan; with sintrom it fell from 3·5 mg.% to 0·7 mg.% in the same time. After dicoumarol, however, the blood concentration fell much more slowly, and an initial level of 4·8 mg.% reached only

* Geigy Pharmaceuticals, Basle, kindly supplied Sintrom for use in this investigation.

2·5 mg.% after 2 hours and was still 2·0 mg.% after 6 hours.

Pratt[7] found that sintrom had a very low toxicity. Rabbits withstood dosages of 0·5 - 5 mg./kg. for 2 months without symptoms. Animals whose prothrombin level was maintained at 20-40% for 9-12 weeks, showed no post-mortem internal bleeding or toxic liver damage. The haemoglobin concentration and blood picture of these animals remained unchanged.

In an animal experiment to show the lethal dose of various antithrombic substances, Montigel and Pulver[6] found that 1,470 mg./kg. of sintrom was lethal to a mouse, as opposed to 840 mg./kg. of tromexan.

Jürgens[5] claims that the action of sintrom begins within 12 hours after administration of a single dose, and reaches its maximum effect after about 2 days. It remains at this level for a brief period, followed within the next 2-3 days by a relatively rapid complete normalization.

Aeppli and Rubeli[14] state that the maximum anticoagulant effect of sintrom occurs as rapidly as with tromexan, but remains constant for 15-20 hours, after which the prothrombin level rises rapidly.

To assign sintrom a place in the anticoagulant drug 'spectrum', it has been suggested that it is intermediate in action between tromexan and dicoumarol.[6,10,20] Tromexan has a short transient action and a relatively low activity and toxicity; dicoumarol acts for a longer time and has a higher activity and relatively high toxicity.[6]

MATERIALS AND METHODS

Eighteen patients (Table I) having various thrombophlebitic disorders of the lower limbs were studied over a period of from 16 to 241 days. The total patient-days were 1,128. All the patients were ambulatory after the initial pain was relieved.

Venous blood samples were collected in the morning and the prothrombin indices estimated within 2-3 hours of collection.

The prothrombin index was determined according to the method of Stein.[11] The results were expressed as 'prothrombin index' (PI), which is calculated by dividing the prothrombin time of normal plasma (estimated daily) by that of the patient's plasma and expressing the result as a percentage.*

Once the maintenance dose had been established, the prothrombin index was estimated once or twice weekly (usually only once a week). We should have preferred to perform the PI estimation more frequently, but this was impracticable, for in many instances the patients lived as far afield as 40 miles.

Clinical examinations were carried out on every patient before and during the investigation. Particular attention was paid to the duration of bleeding from the venepuncture site, when blood had been withdrawn for PI estimations.

RESULTS AND DISCUSSION

Administration of Dosage

Sintrom is supplied in 4 mg. tablets. Neill *et al*[3] found that 2 mg. of sintrom are roughly equivalent to 25 mg. of dicoumarol (i.e., one 4 mg. tablet of sintrom = 50 mg. of dicoumarol) by using the following method: 35 patients were initially treated with dicoumarol and when the average maintenace dose of each patient had been established, sintrom was substituted to maintain an equivalence in dosage between these two drugs.

The mean *induction dose* for sintrom in this series was 34·4 mg. (8·6 tablets). The range was from 6 to 12 tablets, given in either divided or single doses. Neill *et al.*[3] used 2 induction doses on successive days and Menéndez *et al.*[8] recommended an induction dose of 32 mg. given in a single dose. We did not find that either the divided or single induction dose method had any advantage over the other. Jürgens[5] found that the rapidity of onset of action is practically independent of whether sintrom is given at one time or in fractionated doses.

In an attempt to verify these conclusions further, we performed the following experiment: 2 normal subjects

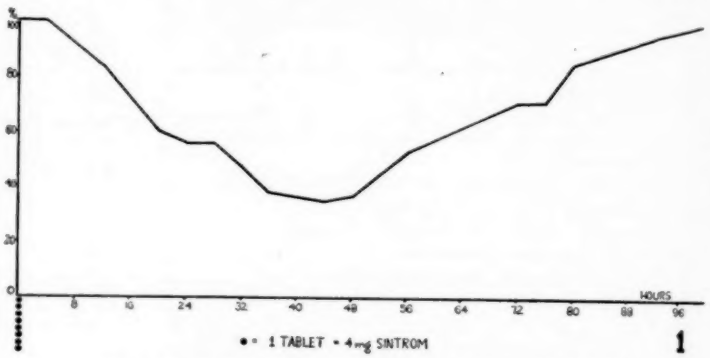


Fig. 1. Single dose of sintrom administered to normal subject A.

(A and B) were given single and divided doses respectively. A, weighing 150 lb., received 32 mg. in one dose. B, weighing 135 lb., received 16 mg. and, 24 hours later, another 12 mg. PI estimations were performed at 4 hourly intervals, whenever possible, by the finger-prick method of Stein and Wallace.[19] A decided fall in the prothrombin index was obvious 12 hours after the induction dose in both cases. The lowest point was reached in 44 and 36 hours respectively. The prothrombin index remained fairly constant for a short while, after which

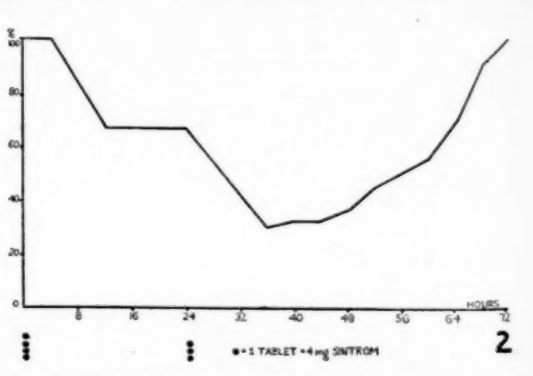


Fig. 2. Divided dose of sintrom administered to normal subject B.

* These results can be converted to prothrombin concentration or coagulation valency by means of a calibration curve.[11]

TABLE I. SUMMARY OF 18 CASES TREATED WITH SINTROM

Sex	Age	Weight (lb.)	Disease	No. of days on treatment	Dose: Induction (a) (tablets)	Dose: Average maintenance (mg.)	% of days within therapeutic range (30-70%)
Male	28	150	Acute deep-vein thrombophlebitis in a post-phlebitic leg	129	8	5·0	68%
Male	34	200	Deep-vein thrombophlebitis in a post-phlebitic leg	20	7	3·7	92%
Female	44	155	Deep-vein thrombophlebitis	50	7	5·0	41%
Female	24	130	Deep-vein thrombophlebitis	28	7	7·0	43%
Female	39	155	Deep-vein thrombophlebitis	51	6, 4	5·6	62%
Female	40	145	Deep-vein thrombophlebitis	92	2, 2, 2	5·4	21% (d)
Male	65	160	Deep-vein thrombophlebitis plus infectious mononucleosis	59	4, 3	2·5	89%
Female	21	105	Deep-vein thrombophlebitis plus infectious mononucleosis	55	heparin	2·5	54%
Female	42	180	Recurrence of deep-vein thrombophlebitis in a post-phlebitic leg.	96	4, 4	3·9	89%
Female	47	162	Post-operative (radical for varicose veins) thrombophlebitis	51	dicoumarol (b)	3·8	77%
Male	44	160	Post-operative (radical for varicose veins) thrombophlebitis	30	8	4·3	100%
Female	62	200	Acute deep and superficial thrombophlebitis in a post-phlebitic leg	54	8, 4	4·9	71%
Female	70	95	Acute superficial thrombophlebitis	54	7, 4	4·1	95%
Female	56	186	Superficial thrombophlebitis.	17	8, 4	5·8	85%
Female	37	280	Superficial thrombophlebitis	16	7	4·0	81%
Male	52	150	Superficial thrombophlebitis	36	heparin	5·4	29% (d)
Male	47	200	Extensive thrombophlebitis migrans.	241	7, 4 (c)	4·0	86%
Male	42	170	Idiopathic deep-vein thrombophlebitis plus arterial spasm.	49	8	3·8	100%

(a) Where 2 (or 3) figures are given, the induction was by means of doses given on 2 (or 3) successive days.
(b) On dicoumarol treatment initially.
(c) On dicoumarol treatment initially but allowed to return to normal before sintrom administration.
(d) Patients who were not cooperative.

TABLE II. PHARMACOLOGICAL PROPERTIES

Chemical Name	Name of Drug	Time required to reach therapeutic levels (hours)	Time required to return to normal after single dose (days)	Dosage: Initial (mg.)	Dosage: Maintenance (mg.)	Side-effects
3, 3'-methylene bis(4-hydroxycoumarin)	Dicoumarol Dicoumarin	48-72	5-6	200-300	25 or less—100	Nausea, depression, nightmares
bis-3,3'-(4-oxycoumarinyl)-ethyl-acetate (B.O.E.A.)	Tromexan	18-36	1-2	1,200-1,500	150-900	Unknown in our experience
3-[-(4'-nitrophenyl)-β-acetyl-ethyl]-4-hydroxycoumarin	Sintrom	24-72	2-3	32	2-7	No side-effects
3(α-acetonyl benzyl)-4-hydroxycoumarin	Sodium Warfarin (Coumadin)	24-36	5-6	50-75	5-10	Unknown in our experience
phenyl-indanedione	Dindevan Indema	24-36	2-3	150-300	25-100	Gastro-intestinal upset and nausea
2-(diphenyl-acetyl)indane- 1:3-dione	Dipaxin	24-72	up to 6	20-30	2-5	Unknown in our experience
2-diphenyl-acetyl-1,3-indanedione	Didandin (Diphenadione)	48-72	15-20	20-30	5-10	Unknown in our experience

there was a rapid return to normal (in 2 and 1 days respectively). (Figs. 1 and 2.)

Sintrom induces a therapeutic hypoprothrombinaemia in most patients 36 hours after the initial dose is given.[3] Neill *et al.*[3] found that 94% of their patients were in the therapeutic range by the end of 43 hours after the induction dose. We found that 77% of our patients were within the therapeutic range (PI 30-70%, corresponding to a coagulation valency of 10-42%) in 24 hours. All the patients except one who did not come for his PI estimation on the appointed day) were therapeutically controlled in 48 hours.

The average *maintenance dosage* in our series varied from 2·5 to 7 mg. (mean=4·5 mg.) daily as indicated by the prothrombin index. Relatively small changes in the dosage rapidly influenced the prothrombin index. The doses were adjusted as follows:

Prothrombin Index	*Daily Dose of Sintrom*
30-40%	2 mg. (=½ tablet)
40-55%	4 mg. (=1 tablet)
55-70%	8 mg. (=2 tablets)
over 80%	10-12 mg. (=2½-3 tablets)

The maintenance doses as well as the initial doses were administered once daily, reducing thereby the cost and inconvenience of the treatment. This is an advantage sintrom has over other quick-acting coumarin derivatives, which must be given several times a day to obtain a constant prothrombin index.

The maintenance dose, once established, did not vary greatly, although it was sometimes found that a patient who had been well controlled for several weeks on 1 tablet daily, suddenly required more (i.e. 2 tablets daily). The daily dosages did not usually differ greatly from patient to patient (Table I), although Neill *et al.*[3] found that there was a wide variation from patient to patient in the maintenance doses. Out of a total of 100 patients they found that 29 required as little as 2-4 mg. daily or as much as 10-12 mg. daily.

The effects produced by sintrom, as with all anticoagulants of the coumarin type, can be reversed by the administration of vitamin K_1.[8,3] Vitamin K_1 need only be used in cases of severe haemorrhage, because it acts very rapidly as an antidote to sintrom.[8] Neill had 2 patients whose prothrombin activities were below 10%. Twenty hours after intravenous injections of vitamin K_1, the levels rose by 64% and 40% respectively.[3] In mild haemorrhages, it is sufficient merely to interrupt the dosage in order to stop the bleeding.[8] This may be regarded as a practical advantage, because the action of vitamin K_1 complicates subsequent therapy with

coumarin derivatives.[6] Should surgery or a tooth extraction become necessary during therapy, it is a simple matter to allow the prothrombin index to return to normal within a short while (2-3 days) by cessation of therapy. Neill *et al.*[3] found that a patient whose prothrombin index had been depressed to a hazardous level could be brought to a safe therapeutic level by eliminating one dose.

Side-effects. Sintrom is well-tolerated orally and has no side-effects in the form of nausea, anorexia, headache or vertigo, and has a low toxicity. In their animal experiments Montigel and Pulver[6] found that the lethal dose of sintrom administered to mice is much higher even than that of tromexan. Two of our patients, who were on dicoumarol therapy initially and subsequently continued with sintrom, were very favourably impressed by the sense of well-being they experienced on the change of drug. Some of the other anticoagulant drugs, dicoumarol especially, may cause severe mental depression, irritability, and sleeping irregularities in the form of nightmares. Sintrom, so far, has produced no such side-effects.

Some of the pharmacological properties of sintrom are compared with those of other coumarin and indanedione anticoagulants in Table II.

Contra-indications. As with other coumarin derivatives, sintrom should not be used in the presence of haemorrhagic diathesis, severe parenchymal liver damage, kidney insufficiency, ulceration of the digestive tract, or pregnancy, and during neurosurgical interventions. Care should be exercised if sintrom is administered together with other drugs, because salicylates[15] and PAS, for example, sometimes diminish the prothrombin index, while digitalin and strophantin increase it.[16]

Clinical Management of Cases

The patients investigated in this series varied in age from 21 to 70 years and suffered from thrombophlebitis of one type or another in the lower limbs:

1. Acute deep-vein thrombophlebitis: femoral, iliofemoral and those presenting in the deep venous plexus of the calf.
2. Superficial thrombophlebitis: (*a*) due to trauma, (*b*) secondary to varicose veins, (*c*) idiopathic superficial thrombophlebitis migrans, and (*d*) localized superficial idiopathic thrombophlebitis.
3. Recurrent acute thrombophlebitis in a post-phlebitic limb.

It is now our custom to use anticoagulant therapy in all cases of thrombophlebitis—superficial and deep. The reason for this is that, although thrombo-embolic phenomena are commoner following deep thrombophlebitis, cases of thrombo-embolism following superficial thrombophlebitis have been reported.[15] In the experience of one of us (I.N.) two such embolic phenomena resulted from a superficial thrombophlebitis before the use of routine anticoagulant therapy. As soon as the acute pain had subsided, the patients were made ambulatory and supplied with efficient supportive bandaging. Elevation of the bed when sleeping and brisk walking rather than standing were insisted upon, as well as elevation of the leg when sitting. Smoking was not allowed and the obese patient was ordered to reduce weight.

Anticoagulant therapy was usually recommended for at least 8 weeks after the pain had subsided, because of the high incidence of recurrences with insufficient therapy.

Haemorrhages, nose bleeds and macroscopic blood in the urine did not occur, although prolongation of the menstrual bleeding occurred in some of the female patients.

Results

Although most workers consider the therapeutic range for the PI to be between 30 and 50% of normal, corresponding to a coagulation valency of 10-21%,[12] others[13] have aimed at ranges of 50-70% (coagulation valency 21-42%). In this study we have aimed at maintaining the prothrombin index between 30 and 70%. We realize that there may be some objection to this wide range, but since all our patients were ambulatory we feel we were justified. Clinically, we have noticed that there is a marked improvement in the patients' condition even when the PI is 70%. Although

TABLE III. CONTROL OF PROTHROMBIN INDEX

% of Days during which PI between 30-70%	*% of patients*
75-100	56
over 60	72

the figures shown in Table III do not seem to reflect a very good degree of control of the prothrombin index, it should be emphasized that, whereas in practice one is satisfied if the prothrombin index is slightly below or above the therapeutic range, strict interpretation of the results has been employed for the purpose of investigating the action of sintrom. If a patient's prothrombin index was immediately below 30% or immediately above 70%, it was regarded as being out of therapeutic control. Also, some of the patients in this series were difficult to control because of frank or suspected lack of cooperation and because PI estimations could not be performed more frequently than once a week. However, as it was our intention to show that sintrom can be given with safety to the ambulatory patient who is not under medical supervision for a large part of his or her treatment, it was deemed expedient to include these in this report.

Sintrom does not accumulate in the body[6] and the toxicity of the drug is so low that the effects of overstepping the prescribed dosage would not be disastrous. A week's supply of tablets is usually issued to the patient.

In all cases we found that the drug was well tolerated and patients did not suffer from any side-effects. There was no recurrence of thrombophlebitic disorders, or of complicating thrombo-embolic phenomena during therapy in the present series. No case of haemorrhage requiring cessation of

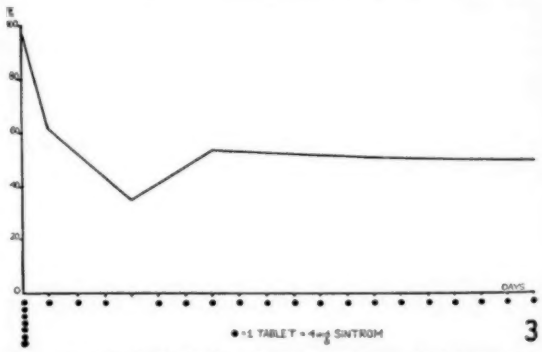


Fig. 3. Single induction dose of sintrom administered to patient.

therapy or the administration of vitamin K_1 occurred. Menéndez *et al.*[8] reported that vitamin K_1 in 50 mg.-intravenous doses is effective as an antidote to any bleeding that may occur.

In the limited study, we found that neither the single nor divided induction dose methods seemed to have any advantage over the other. Figs. 3 and 4 show the degree of control that can be achieved in the ambulatory patient with single and divided induction doses respectively.

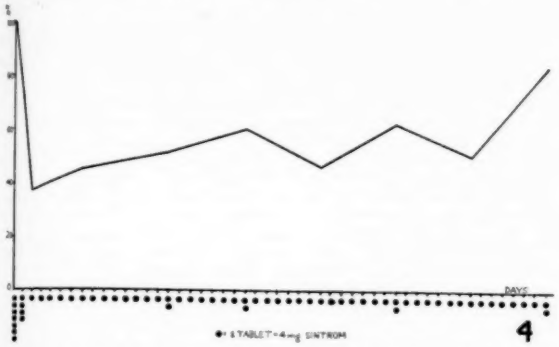


Fig. 4. Divided induction dose of sintrom administered to patient.

It is also possible to change from dicoumarol to sintrom without any difficulty, as can be seen from Fig. 5.

We feel that a further study is necessary, and we hope to present results on hospitalized patients treated with sintrom in a forthcoming publication.

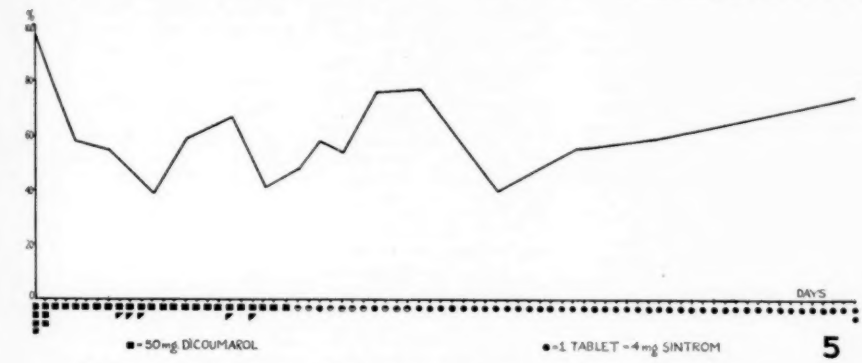


Fig. 5. Patient's therapy initially dicoumarol, subsequently changed to sintrom.

SUMMARY

We have presented a report on our experience in treating 18 ambulatory patients suffering from thrombophlebitis with a new coumarin anticoagulant drug called Sintrom.

1. The chemical and pharmacological properties of the drug are briefly described.

2. Sintrom induces a therapeutic prothrombin level (index 30-70% of normal, corresponding to a coagulation valency of 10-42%) in most patients 24 hours after the initial dose, which is given as a single or divided dose of 24-48 mg.

3. Maintenance doses are given once daily and vary from 2-7 mg. per day.

4. Estimations of the prothrombin index were usually performed once or twice a week, but this is not ideal.

5. No haemorrhages or thrombo-embolic complications occurred.

6. No undesirable side-effects were caused by the drug.

7. After cessation of therapy there is a rapid return to normal (1-2 days in 2 normal subjects).

8. Tentatively, it may be concluded that sintrom is a satisfactory anticoagulant for use in the acute and ambulatory case of thrombophlebitis.

We wish to express our sincere thanks to Dr. B. A. Bradlow and Dr. D. Mendelsohn for their kind suggestions and criticisms.

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AMERICAN MEDICAL ASSOCIATION, COMMITTEE ON INJURY IN SPORTS

This Committee has condemned crash diets, sometimes approaching the starvation level, by wrestlers, boxers and footballers trying to make a certain weight class. 'Under the strong motivation and appeal of sports the diets and drying out may be carried to great extremes. Such efforts are not consistent with the spirit of sport in that they tend to defeat regulations designed to insure fair and equitable competition. Disturbing the fluid balance of the body by drying out holds serious health hazards; these dangers are intensified in the immature organism of the growing adolescent athlete. They are also intensified by periodic weighings which encourage the athlete to resort to such practices at frequent intervals during the season'.

THYROID FUNCTION IN RELATION TO BILE LIPIDS AND BILE ACIDS*

AN EXPERIMENTAL APPROACH BY CANNULATION OF THE BILE DUCT OF RATS SO THAT THE ENTERO-HEPATIC CIRCULATION IS MAINTAINED

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It is known that the main metabolic end-products of cholesterol in rats are cholic acid and deoxycholic acid, and that rats with biliary fistulae secrete these bile acids as their taurine conjugates, whereas most faecal bile acids are unconjugated. In rabbits and man, however, deoxycholic acid is formed from cholic acid during the entero-hepatic circulation and appears in bile together with cholic acid and chenodeoxycholic acid.[3] In rats treated with thyroid hormone the cholic acid in bile decreases rapidly,[5] whereas the bile cholesterol and chenodeoxycholic acid secretions are increased.[2]

A great deal of work on bile acids has been performed in rats with biliary fistulae, and consequently, with no entero-hepatic circulation. Moreover, most methods for quantitative bile-acid analysis measure trihydroxycholanic and dihydroxycholanic acids separately, but do not differentiate between chenodeoxycholic acid and deoxycholic acid. Under these conditions, and also because of the absence of a gall-bladder in the rat, it would be impossible to demonstrate the presence of deoxycholic acid in the rat, even if it were formed from cholic acid by the action of intestinal micro-organisms as has been suggested is the case in man and the rabbit.

The purpose of the present investigation was, firstly, to develop a technique for bile-duct cannulation in rats in such a way that the entero-hepatic circulation was maintained; secondly, to devise a simple method for the routine quantitative analysis of deoxycholic acid and, thirdly, to compare the influence of thyroid hormones on the bile volume, bile lipids and bile acids of rats with biliary fistulae as against those of rats in which the bile re-circulates.

In rats whose entero-hepatic circulation was maintained, 2 cannulae were inserted into the common bile duct—one into the peripheral end and the other into the central end. The cannulae were connected on the backs of the rats, allowing free movement of the animals. Bile was collected for 6 hours every 2nd day and was compared with bile from rats with continuous biliary drainage as described earlier.[5] Bile cholesterol, lipid phosphorus, cholic acid and deoxycholic acid were measured in thyroidectomized, normal, and hyperthyroid rats. For the analysis of deoxycholic acid the ultra-violet absorption technique of Mosbach[4] was modified and applied to unhydrolyzed bile. The following equation was derived for the estimation of the 'true' bile cholic and deoxycholic acid:

$$x = 153{\cdot}39\ E_1 - 28{\cdot}00\ E_2$$
$$y = -32{\cdot}78\ E_1 + 111{\cdot}70\ E_2$$

where x = pure cholic acid; y = pure deoxycholic acid; E_1 = optical density measured at 320 mμ after 15 minutes of incubation at 60°C with 65% H_2SO_4; E_2 = optical density measured at 385 mμ after 60 minutes of incubation at 60°C with 65% H_2SO_4. The mean recovery for this method on unhydrolyzed bile was 80% (range 71-88%) for cholic acid, and 82% (range 72-90%) for deoxycholic acid.

Bile samples (6 hourly every 2nd day) of thyroidectomized rats in which the entero-hepatic circulation was maintained, were pooled; part of the bile was hydrolyzed, incubated with H_2SO_4 and the absorption spectra (200-440 mμ) of the unhydrolyzed bile samples compared with those of hydrolyzed bile. The results were then compared with those obtained from rats treated with tri-iodothyronine (20 μg. T_3 per day). A decrease of 30% in the cholic-acid concentration was observed in the bile of thyroidectomized rats during hydrolysis, whereas the dihydroxycholanic acids as measured at 385 mμ, increased by 59% in the bile of both thyroidectomized rats and rats treated with tri-iodothyronine during hydrolysis of the bile. The drop in cholic acid concentration, as affected by hydrolysis, is ascribed partly to the liberation of taurine from chenodeoxycholic acid, and partly to the destruction of cholic acid by alkaline hydrolysis. The increase in dihydroxycholanic acids during hydrolysis is due to the shift of the free chenodeoxycholic acid peak from 305 mμ (for taurochenodeoxycholic acid) to 385 mμ (for free chenodeoxycholic acid). At the same time hydrolysis causes a shift of the taurodeoxycholic acid peak at 389 mμ to that of free dihydroxycholanic acids at 385 mμ demonstrating the existence of taurodeoxycholic acid in the unhydrolyzed bile of rats in which the entero-hepatic circulation is maintained. In the bile of rats treated with tri-iodothyronine hydrolysis causes a shift of the cholic acid peak at 320 mμ to that of 350 mμ, thereby demonstrating the decrease of cholic acid and the simultaneous increase in taurochenodeoxycholic acid in hyperthyroidism.

In group studies on rats it was demonstrated that treatment with tri-iodothyronine and tri-iodothyroacetic acid produced an increase in concentration of bile cholesterol, lipid phosphorus, chenodeoxycholic acid and deoxycholic acid as well as an increase in their respective quantities secreted for 6 hours every 2nd day. The bile concentration and total output of deoxycholic acid were increased to a greater extent the longer the bile was allowed to re-circulate. In every case the cholic acid concentration decreased by at least 1/3rd of its original value during treatment with thyroid hormones. It was possible to demonstrate these changes repeatedly on the same thyroidectomized rats with bile in re-circulation in experiments lasting as long as 1 month. The volume of bile secreted was markedly increased by treatment with thyroid hormone.

On the basis of these findings it is postulated that thyroid hormone stimulates the whole metabolic pathway from acetate to cholesterol, and all the catabolic pathways of cholesterol to bile acids and ketocholanic acids. In rats in which the entero-hepatic circulation is maintained, such stimulation will result in the decrease of cholic acid and the increase of chenodeoxycholic acid and deoxycholic acid, as follows:

1. Chenodeoxycholic acid is formed from cholesterol but, since the resulting chenodeoxycholic acid is not a precursor of cholic acid, it will accumulate in bile.
2. Deoxycholic acid is formed from cholesterol as an intermediate stage in the formation of cholic acid. If, however, the entero-hepatic circulation is maintained, the cholic acid is reconverted back to deoxycholic acid. Part of the remaining cholic acid is further metabolized to ketocholanic acids.
3. Lithocholic acid is formed from cholesterol which, in rats, may be changed to chenodeoxycholic acid.
4. Chenodeoxycholic acid is formed from 3α, 7α-dihydroxycoprostane and cholic acid from 3α, 7α, 12α-trihydroxycoprostane. The cholic acid is again either converted to dihydroxycholanic acids, or further oxidized to ketocholanic acids.

All these reactions will lead to the disappearance of cholic acid and the accumulation of chenodeoxycholic and deoxycholic acids in bile when the thyroid is hyperactive. The reverse, i.e., the increase of cholic acid in hypothyroid rats, will inhibit cholesterol synthesis.[1]

Since the blood-cholesterol and lipid-phosphorus concentrations are inversely related to the bile-cholesterol and lipid-phosphorus secretion in hypo- and hyperthyroidism, the blood lipids appear to be controlled to a greater extent by endocrine factors affecting mobilization and water metabolism than by metabolic synthesis as influenced by the thyroid.

* Abstract of paper presented at Research Forum, University of Cape Town, 5 May 1959.

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RESEARCH FORUM, UNIVERSITY OF CAPE TOWN

A meeting of Research Forum will be held on Tuesday 4 August at 12 noon in the Bennie de Wet Lecture Theatre, A-floor, Groote Schuur Hospital, Observatory, Cape. Prof. L. Eales will speak on 'Urine and stool investigations in the differentiation of the porphyrias as seen in the 3 racial groups in Cape Town'. All who are interested are invited to attend this meeting.

Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

DIE SEREBROSPINALE VOG

Hoewel Corning in 1885 die prosedure van lumbale punksie uitgevoer het by die behandeling van 'n geval met tuberkuleuse meningitis, is die kliniese gebruik daarvan te danke aan die werk van Quincke in Duitsland en Essex Wynter in Engeland wat dit in 1891 by die behandeling van hidrosefalus gebruik het. Fürbinger in 1895 en Netter in 1898 wys op die diagnostiese waarde van makroskopiese veranderings in die serebrospinale vog (S.S.V.), en het ook die chemiese norme van sy proteïen- en chloried-inhoud in normale en patologiese toestande opgestel. Die ontwikkeling tot 1908 van die Wassermann-reaksie en, in 1912, die kolloïdale goudpresipitasie van Lange, leen hulle tot diagnostiese gebruik op die S.S.V. Mestrezat se ekstensiewe studies oor die normale en patologiese chemie van die S.S.V. lui dan die era van wetenskaplike studie van S.S.V.-veranderings in.[1]

Die klassieke werk van Greenfield en Carmichael,[1] hoewel vanjaar 34 jaar oud, is nog steeds die standaardwerk oor S.S.V.-veranderings op fisiologiese en patologiese gebied.[3] Dit moet egter nie beskou word as 'n teken van stilstand in die navorsing in hierdie vak nie. 'n Onlangse simposium[2] getuig van ywerige werk op hierdie gebied.

Isotoopstudies toon dat die sirkulasie van die vog, met nie-belangrike onakkuraathede, voldoen aan die klassieke konsep waarmee ons vertroud is, en wat in 1914 deur Weed beskryf is.[2] Tot dusver is die belangrikste bydrae van die nuwer isotoopstudies die klem op die dinamiese status van die komponente van die vog en 'n veelvuldige saamvoeging en wysiging van die saamstelling daarvan deur die choroïdpleksusse, ependiemselle en serebrale perivaskulêre ruimtes, asook deur die vate in die subarachnoïede ruimte.[2]

Lumbale punksie sal miskien meer dikwels in die algemene praktyk uitgevoer word as daar besef word dat dit 'n valse redenasie is dat die vog nie ondersoek sou kon word nie. Baie kan geleer word uit net 'n makroskopiese ondersoek.[1] *Troebelheid* dui op die teenwoordigheid van selle of organismes en is soms aan fibrien te wyte. As die vog 'n rukkie staan mag 'n fibrien-stolsel sigbaar word wat *nie* diagnosties is van tuberkuleuse meningitis nie, aangesien dit ook in poliomielitis en neurosifilis gevind word. Xantokromie is altyd patologies en kan te wyte wees aan subarachnoïede bloeding of 'n hoë proteïen-gehalte (bv. Froin se sindroom). Die teenwoordigheid van waarneembare bloed in die S.S.V. dui op 'n subarachnoïede bloeding of deurbraak na die ventrikels of subarachnoïede ruimte in gevalle met intraserebrale bloeding.[1] 'n Lumbale punksie mag soms die enigste wyse van differensiasie tussen 'n meningitis en 'n subarachnoïede bloeding wees.[4]

Eenvoudige toetse soos dié van Pandy en 'n gewysigde Benedict-toets kan 'n growwe kwantitatiewe idee van die proteïen- en suiker-gehalte gee, en vereis net die reagens en 'n proefbuis.

Die selinhoud van normale vog is baie konstant en word gestel op drie limfosiete per c.mm. as die bogrens van normaal. Geen polimorf behoort in normale vog gevind te word nie, en vier limfosiete per c.mm. moet met agterdog bejeën word.[1]

Die selinhoud van abnormale vog kan verdeel word in: (1) *Eenkernige selle* (limfosiete), (2) *gemengde selle* (limfosiete en polimorfe), en (3) *polimorfe pleositose*. Laasgenoemde dui op die bevinding van ongeveer 75 persent polimorfe, aangesien daar gewoonlik ± 10 persent limfosiete is.[1] Die verhoging van die aantal selle kan gering, matig, erg of uitgesproke wees (5-10, 10-50, 50-250 en 250+ per c.mm. respektiewelik).

Die klassieke beskrywings soos in verskillende siektetoestande gevind, regverdig nie dogmatiese stellings nie, en Locoge en Cumings[3] toon dat daar 'n groter variasie bestaan as wat ons geneig is om te dink. Uit 12,000 ondersoeke van serebrospinale vog oor 'n tydperk van 14 jaar vind hulle uit 835 gevalle van tumor 'n selverhoging in 8·3 persent en 'n verhoogde proteïen in 71·1 persent. 6·1 persent epilepsielyers toon 'n proteïen bo 70 mg. persent en twee gevalle het meer as 100 mg. proteïen getoon. Die bevindings by spinale tumore is soos verwag, maar proteïen-waardes tot 4,000 mg. is gevind.

Die bevindings in 770 gevalle met neurosifilis was onverwags. Daar is 'n relatiewe hoë aantal negatiewe Wassermann-reaksies in die bloed sowel as in die S.S.V. gevind in meningovaskulêre sifilis en definitief meer as wat verwag is (8 persent) in dementia paralytica. In 8 persent van gevalle was die Wassermann-reaksie van die bloed positief, maar dié van die S.S.V. negatief by gevalle met dementia paralytica en tabes dorsalis.

'n Uitgebreide literatuur het sedert die monogram van Greenfield en Carmichael ontstaan. Nuwe toetse word op die S.S.V. gedoen, soos transaminase- en ander ensiembepalings, elektroforese van proteïen, isotoopstudies, ens.

Ons is nie bekend met die mate van variasie van bevindings in algemene gestelsiektes nie. 'n Uitgawe soos die Ciba-simposium[2] gee 'n indruk van die omvang van navorsingswerk op hierdie gebied, maar die wenslikheid van 'n samevatting van die *kliniese* toepasbare bepalings en bevindings van die navorsers word allerweë gevoel.

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YOUTH AND AN OUTSIDE INTEREST GROUP

No doctor can dissociate himself from the social tendencies that shape and determine the behaviour patterns in the community which he serves. The fact, therefore, that violence has become such an important element in the numerous

influences to which the teenager is exposed must be of some significance to the doctor who is aware of the broader obligations of his calling.

The teenager, today, is confronted with the insidious influence of radio, cinema, comics, gramophone records and television, all of which are often based on an idiom of violence. It is important to realize that the cumulative influence of these media is producing a destructive effect on the psychological balance of the teenager. Many young persons, today, lack the moral, emotional and intellectual equipment which would enable them to withstand the pernicious effect of the endless stream of violence, crime and brutality to which they are constantly being exposed. This discrepancy between the teenager's moral equipment with which he has to face the world and the terrifying range of the undermining influences to which he is constantly being subjected is largely responsible for the so-called teenage problem in modern society.

There is little point in salving the conscience of the public by emphasizing the moral supposed to be employed in most of the media to which the teenager is exposed—that the 'bad man' will ultimately be vanquished—for, unless the child has been conditioned from an early age to moral values and traditional virtues, he will find the appeal of the 'bad man' far more exciting and in keeping with his own budding aggression than the thin triumph of virtue.

A child brought up in a home with immature and temperamentally unstable parents is particularly vulnerable to these influences, especially at puberty, when sexual maturity produces a desire for independence and originality. Egotistic, aggressive and rebellious traits appear, usually directed against the parents, and emotions tend to be explosive. A special vocabulary is cultivated, and gaudy and conspicuous apparel worn, tendencies which stem from the powerful urge to find a new group to which his loyalties can be attached.

The teenage mind seeks adventure and thrills, but above all it seeks a group to which it can belong. These psychological and functional changes are the natural accompaniment of sexual maturity, and it is at this time that the mind of the teenager seeks an 'outside interest group'. Unfortunately society has failed to provide adequate facilities of this nature. Society has made no attempt to provide such an 'outside interest group' which could satisfactorily direct teenage loyalties, emotional aspirations and ideals. The inevitable consequence of this failure has been the teenager's attempt to find his own substitutes for the 'outside interest group' which he so urgently needs, substitutes which are often undesirable, undermining and anti-social.

It is from this environment that delinquency stems. Freedom, with its rules of behaviour, is replaced by licence. Usually those people who set the pattern for teenage behaviour and who become the heroes of the teenagers, exploit this situation for the benefit of box-office receipts. Because the cinema, television and recording artists present this licence to the teenager *without disapproval*, licence is mistaken for freedom and becomes the cherished goal of many a frustrated young person. Moreover, there are many parents today who seem incapable of distinguishing between adolescent freedom and complete licence. Adolescent freedom should be encouraged as the natural outcome of normal emancipation from parental protection, but destructive licence should be seen for what it really is: a gross deception of oneself.

The period of instability usually starts at puberty, when adolescent crime, too, manifests itself. Children who have a satisfactory 'outside interest group' at school, sometimes experience a period of emotional instability after leaving school. Their lack of experience in coping with new emergencies now begins to tell. At this time there is a great need to belong somewhere, to be recognized, wanted and loved. Moodiness is frequent and feelings of intense loneliness quite common.

It is at this stage, too, that the demoralizing influence of 'rock and roll' stars play havoc with the vulnerable and susceptible teenager. Adolescents have a need for rhythmic expression and it is understandable that this primitive type of music finds a fertile field of response in the teenager. This is only one of the many emotional needs which should be fulfilled by the establishment of a satisfactory 'outside interest group', the only aim of which should be to serve the true interests of the teenager and not to exploit his vulnerability for commercial purposes.

It is vitally necessary for society to recognize that, in the face of some of the decadent influences prevalent today, the community must provide a broad and healthy 'outside interest group' to assist the teenager in his normal development towards emancipation, and at the same time provide positive cultural values to counteract the morally-undermining effect of present-day entertainment media.

Communal youth organizations, financed by the public, should provide facilities for recreation, entertainment and cultural activities in which the teenager can find a healthy outlet for self-expression and in which he can learn the true value of freedom as a social heritage.

SOME CONTRIBUTIONS TO SURGERY OF THE HAND*

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I make no apology for discussing a subject as humble as the hands; much original thought and genuine progress have come from the application of sincere study and accurate observations on the meanest problems. Moreover, because of its importance to mankind, the hand has attracted the attention of surgeons throughout the years.

* Paper presented at the combined Surgical Forum of the Department of Surgery, University of the Witwatersrand, and the Association of Surgeons of South Africa (Johannesburg Sub-group) on 8 July 1958, and at a symposium on Hand Surgery, Rehabilitation Association, Brenthurst Clinic, Johannesburg, on 15 October 1958.

Johannesburg—and the University of the Witwatersrand—has reason to be proud of its contributions to the anatomy and surgery of the hand. Four theses have been written by its sons, namely, Mr. Jack Allen, Mr. Trevor Jones, Mr. H. Gaylis and myself. Mr. Lee McGregor's *Synopsis of Anatomy* has further contributed to the anatomy of the hand.

Much of the work to be discussed in this paper was done 18 years ago. At that time, Kanavel and Shaw's valuable contributions on the hand had been published almost as long again before.

The character of an animal is reflected in the structure of the

upper limb. The activities of civilized man and his pursuits as workman or artist are reflected in the close brain-eye-hand correlation that is man's chief characteristic. The distal articular surface of the metacarpo-phalangeal joint, in particular, is different in man as compared with apes; so, too, are the metacarpal heads. Apart from the more powerful opposable thumb of man, the main differences between the hand of man and ape are to be seen in the metacarpo-phalangeal joints. The human being has more side movement in flexion than the limited up-and-down movement in apes, to favour thumb-index sensory and motor approximation and the formation of a fist.

It behoves surgeons to be interested in hand surgery. Today there is a vast amount of industrial W.C.A. hand surgery that requires treatment according to the highest tenets of our profession. It is only through the surgical teachers that basic knowledge concerning hand surgery is handed on to our students. Hand surgery has become a casualty problem, and surgeons who practised in the 'golden era' of hand surgery, when at least 20% of hospital beds in surgical wards were occupied by hand cases, must realize that a low ebb has been reached in the teaching of hand surgery.

Hand surgery is the meeting-place for many branches of medicine. Here the general practitioner, general surgeon, orthopaedic surgeon and plastic surgeon can show a common interest; and the physician, neurologist and dermatologist are not excluded.

In this paper I shall discuss why a hand goes wrong under treatment, and how we can endeavour to put it right.

THE FIXED CLAW HAND

In this type of case (Fig. 1) there is practically no movement at the interphalangeal (IP) joints, the wrist is flexed and possibly fixed, the thumb adducted, the metacarpo-phalangeal (MP) joints extended. There is wasting, deformity and severe loss of function. The only help 18 years ago was from Kanavel, who showed in 1929 that a hand allowed to be in a bad position develops a fixed claw hand, whereas a hand in the position of function recovers.

Fig. 1. Fixed claw hand following carpal fracture.

He said that in infections the invasion spreads up the lumbrical canals, round the capsule, and that this causes a fixed claw hand. Apart from infection, however, this condition is seen in burns where only skin is involved, and in immobilized hands. For instance, it has been seen after a wrist dislocation, in severe crippling pain after herpes zoster, and in radial palsy, single extensor tendon injuries and other lesions not related in any way to infection.

Intrinsic Muscles on Movement Vary in Position and Function

Studying the MP joints, let us note that the distal articular surface is an extended structure consisting not only of the distal bony articular surface but also of a thickening of the volar capsules—the palmar ligament. This receives on its sides a special portion of the collateral ligament and a portion of the insertion of the dorsal interossei. Dissections during anatomical courses do not involve study of this view of the anatomy, and much is lost thereby. (See Fig. 2, A and B.)

This extended distal articular surface glides over the metacarpal head from extension to flexion and, by reason of the direct and indirect attachment to it of the dorsal and volar interossei, the line of axis of the muscles and the functional activities vary according to the particular position at the time.

In extension the dorsal interossei can abduct, but otherwise they tend to pull the articular surfaces together and jam the palmar ligament against the metacarpal head.

The volar interossei may actually hyperextend and adduct the fingers, and this is the position usually seen in the late fixed claw hand where the dorsal interossei seem to have wasted through

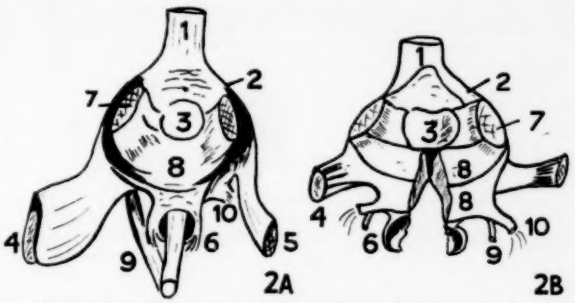


Fig. 2. (A) Right index finger, distal articular structures.
(B) Middle finger distal articular structures.
1, Extensor tendon. 2, Transverse fibres of aponeurosis. 3, Proximal articular surface of phalanx. 4, Dorsal interosseous muscle. 5, Volar interosseous muscle. 6, Fibrous connection to palmar fascia and fibrous flexor sheath. 7, Collateral ligaments. 8, Portion of collateral ligament forming palmar ligament. 9, Lumbrical muscle. 10, Deep transverse palmar ligament.

inactivity, and the volar interossei show a dominance over the dorsal interossei and produce the characteristic deformity.

In flexion the dorsal interossei assist flexion and lateral movements. The volar interossei will also partake in flexion of the MP joints. In extension, maximum contraction will produce minimum movement, whereas in flexion minimum contraction

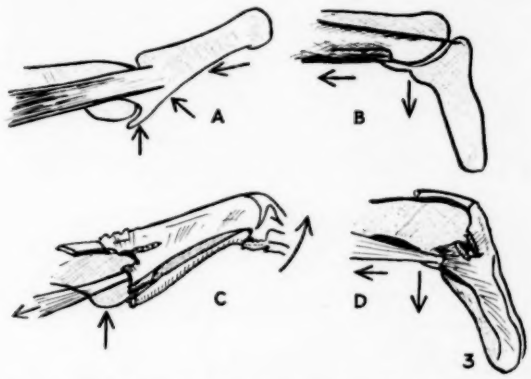


Fig. 3. Changing position of dorsal and volar interossei in extension and flexion.

will permit of maximum movement and assist recovery. This concept of altering anatomy and function in different positions gives a dynamic concept to the study of the hand, with constant variations and changes in mechanics and functions (Fig. 3).

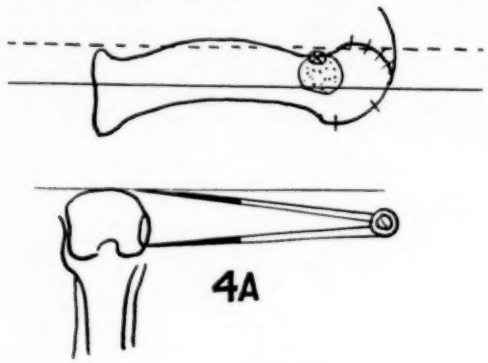
Collateral Ligaments

When there is extension and hyperextension of the MP joints, the dorsal fibres of the collateral ligaments are relaxed, but in flexion the fibres are taut. In the IP joints, the collateral ligament is broad and the head more circular, so that the arc of the fibres is such that with any position of the IP joint some fibres are relaxed. These joints, therefore, may become stiff in any position if kept in that position long enough, whereas it is safe to keep a MP joint flexed as long as one likes. (See Fig. 4, A and B.)

In prolonged immobilization of fingers in extension, especially of the ring or little fingers, a snap will take place from extension to flexion on sustained pressure, and *vice versa*. This is the 'snap phenomenon', due to stretching of the collateral ligament over the most prominent point of the metacarpal head. Microscopically, it is seen in such a case that a bunching up of the fibres of the collateral ligament has happened, and this is seen too in the nuclei. The fibres are wavy instead of straight, the nuclei also wavy and closer together—bunched up, as it were—and corresponding to the clinical state of fixation.

When a MP joint gets stiff from immobilization in extension,

the palmar ligament is jammed against the metacarpal head through the pull of the dorsal interosseous insertions, and attempts to flex the joint are ineffective and only a 'pseudo-flexion' will take place. The dorsal part of the joint may be stretched open, but there is no gliding of the distal articular surface over the head of the metacarpal into flexion.



Fully Developed Fixed Claw Hand

Fixed claw hand is a fully developed deformity dependent on an accumulated disability associated with the effects of wrist drop, prolonged extension of the MP joints, and inadequate movement of the finger joints, as follows:

1. *MP joint extension alone* will result in fixation in that position (collateral ligament shortening), with (*a*) pseudo-flexion (dorsal interosseous palmar ligament insertions), (*b*) snap phenomenon (collateral ligament shortening), (*c*) muscle wasting (disuse atrophy), and deformity (muscle dominance of volar interossei).

The lesion may apply to one finger only, but usually more than one are affected as the neighbouring joints in relation to the pathological one (septic arthritis, metacarpo-phalangeal fracture, single extensor tendon injury, adhesions) also become affected. When the wrist can be extended, and the fingers are therefore strong and the thumb opposed, the disability applies only to the MP joints, but the wrist position is the key to the further and more severe state, when stiff fingers and adduction of the thumb are superadded.

2. *Wrist drop* produces the following developments:

(*a*) It allows extension of the MP joint through pull on the extensor tendons and slack of the flexor tendons and lumbricals, and, even if there is no local pathology, fixation may occur through wrist drop alone, maintained through neglect or continued pain.

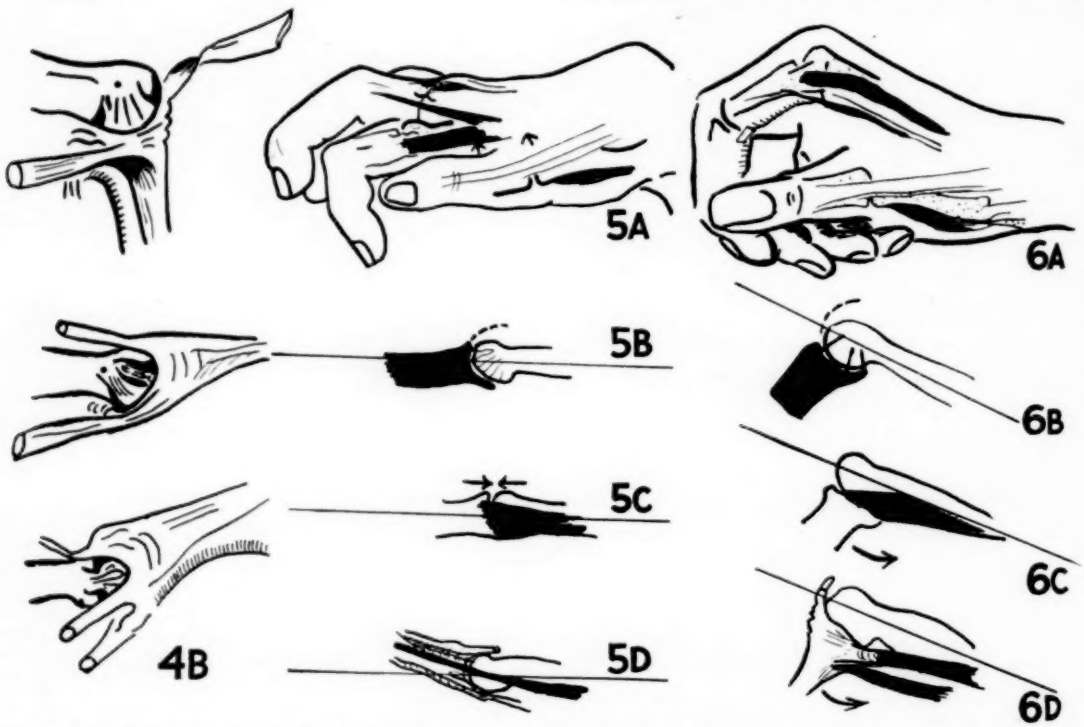


Fig. 4. (A) In the lower figure the callipers measures the distance from the centre of origin of the collateral ligament to a line projected from the articular surface of the head as seen from the side. An imaginary arc is then described (as shown in the upper picture) which shows that the convexity of the articular surface of the head coincides with the lower part of the arc up to the line corresponding to the centre of the long axis of the bone. Thereafter the line of the articular surface falls away within the arc, thus explaining why the collateral ligaments are taut on flexion and relaxed in extension.
(B) Collateral ligaments of MP joint in 3 positions.

Fig. 5. (A) Fixed claw hand.
(B) Relaxation of dorsal fibres of collateral ligament in extension of MP joint.
(C) Dorsal interosseous muscle pulling joint surfaces together in extension.
(D) Volar interosseous muscle hyperextending (and adducting) the MP joint.

Fig. 6. (A) Hand in position of function.
(B) Collateral ligament taut in flexion of MP joint.
(C) Dorsal interosseous muscle in position of mechanical advantage (in flexion of MP joint).
(D) Volar interosseous muscle (and lumbrical). Note changed function of volar interosseous muscle in flexion.

This is seen also in many other lesions that are associated with wrist drop maintained for a long time. The position of ease is not the position of function. Any hand allowed to assume the position of ease, with a supported elbow, will fall into that dreadful position of wrist drop and extended MP joints. And how much worse it is if pain or cumbersome dressings restrict movements as well!

(*b*) Power is lost in the fingers because the flexor tendons are slack. Weak movements at the IP joints without full range will lead to stiffness and eventually fixation of the finger joints.

(*c*) The thumb extends and adducts from extensor tendon pull and slackness of the short thumb muscles of the thenar eminence. When the long flexor of the thumb contracts and the IP joint of the thumb now flexes, the thumb is adducted further, with tightening of the long extensor of the thumb and 'bow-stringing' of the thumb further into adduction.

The picture of the fully developed fixed claw hand is thus one of fixed MP joints in extension, atrophy and deformity, wrist drop, weak, partially-fixed IP joints, and a fixed adducted thumb.

The anatomical explanation of the progression to fixed claw hand is the opposite of that involved in the process of recovery of a hand in the position of function. (See Fig. 5, A, B, C and D, and Fig. 6, A, B, C and D.)

Treatment

MP joints in flexion never get stiff, and early recover movement. Extension of the wrist assists MP joints to flex, gives power to fingers, and allows the thumb to become opposed and to recover in that position.

In a bad hand, one must get the wrist up, and the IP joints moving. In the early stages in a pending claw hand, get the MP joints forward into flexion. It has been said that there is no treatment for a fixed claw hand. This is not true. Dorsal fibres of the MP collateral ligaments can be cut, or the collateral ligaments manipulated by flexing the MP joints, and the hand can be put up in a position of function. This must be maintained for months; and the patient will recover useful hand function.

In the worst hands, start with the MP joints maintained in flexion by the use of plaster of Paris; the IP joint can then use the plaster cross-slab as leverage, with or without elastic traction. The wrist is well extended to give full power to the IP joint movements through the flexor tendons. The thumb is opposed. This position may be maintained for weeks. In one case*, the plaster of Paris was left on for 2-3 months; full recovery took place.

Physiotherapists frown and plead for removal of the plaster of Paris. This is done when recovery is on its way. Wrist drop and mobility certainly aid recovery of extension and the return of MP movements, with the altering position of the wrist, but it is my experience that this will always come if the MP joints have been kept in flexion. They cannot get stiff in this position, unless skin scars or tendon scar to skin is present in the palm.

It is important to recognize the hand in early stages of fixation. If resistance to flexion is encountered at the MP joints, act at once, and manipulate the MP joints into flexion and maintain them so for about a week; the recovery will be dramatic.

FINGER DEFORMITIES

Finger Deformities from Hyperextension of the Proximal Interphalangeal Joints with Flexion of the Distal Joints

This condition may occur from many causes. Kaplan has stated that if the capsule of the joint ruptures hyperextension occurs, but an analysis of cases shows the following:

(*a*) *Physiological.* Some people can flex the distal IP joints while still maintaining the proximal IP joint in extension. This is the reverse of normal, in which flexion occurs first in the proximal joint and the lateral bands 'button-hole' over the sides of the joint to give length to the extensor tendon for subsequent flexion of the distal joint. When the distal joint is flexed first, the lateral bands are tautened into extension, and 'bow-string' the proximal IP joint into hyperextension.

(*b*) *Burns.* In burns, scar under the distal joint will produce this deformity; so will scar over the proximal phalanx, which prevents the lateral bands 'button-holing', so that the distal joint starts to flex.

(*c*) *Dupuytren's Contracture.* This deformity has also been seen in Dupuytren's contracture. What happens is that the proximal IP joint overcomes the scar on the volar surface. In the patient's efforts at extension of the fingers with the MP joints still flexed by the deformity, the intrinsic muscles overact, and the lateral bands pass dorsally. In the continued maintained extension of the proximal IP joint, flexion of the distal IP joints further 'bow-strings' the lateral bands dorsally—hence the deformity. (See Fig. 7.)

Fig. 7. Dupuytren's contracture.

(*d*) *Post-paralytic.* A similar deformity has been seen in a post-paralytic case, and here a weakness of extensors and compensatory over-function of the intrinsics seems to be the basis for the production of an 'intrinsic plus' hand. It is reported that a similar deformity sometimes occurs when the profundus tendon flexes the distal IP joint in the absence of a sublimis to flex the proximal IP joint—but not in my experience. (See Fig. 8.)

Fig. 8. Post-paralytic type of hyperextension deformity of proximal IP joint.

Alternate Immobilization—a new concept

Button-hole deformity is due to loss of function of the central slip of the extensor tendon. The proximal IP joint flexes and the distal joint extends.

* Illustrated at the meeting by a diagram.

The repair of this deformity is surgical, and the results often poor. Simple suture is ineffective or else results in loss of function and stiffness in extension.

There are two functional elements in the aponeurosis, viz. (1) the central extensor mechanism at the proximal IP joint, and (2) the mechanism of the lateral band, which glides separately on movement of the distal IP joint. When an extensor tendon and aponeurosis are sutured, at first the proximal IP joint should be immobilized, while union occurs in the sutured central extensor tendon. This takes approximately 5 weeks, and one must allow freedom of movement in the distal IP joint, to keep the gliding action of the lateral bands. Subsequently, the distal joint should be immobilized and the proximal joint allowed free, so that the lateral bands can move over the sides in the true fashion, and flexion and extension develop at the proximal IP joint. The central extensor tendon develops its function of extending the proximal IP joint in a coordinated fashion in relation to the distal joint.

Alternate finger joint immobilization has a place in the treatment of any injury of the extensor aponeurosis.

Finger Stiffness. A case illustrating the cause of finger stiffness, and the treatment, is worth mentioning. A young lady developed a glomus tumour on the pulp of her right index finger, and it was so painful that after treatment she was seen eventually with a completely stiff finger in extension. There was no other cause than immobilization.

A flap was turned at the proximal IP joint, and the lateral and central slips of the aponeurosis separated at this point, exposing the dorsal fibres of the collateral ligaments, which were then separated and excised from the underlying head of the phalanx. Flexion became possible at this stage, and recovery ensued with return of full function.

For recovery in finger stiffness, the collateral ligaments must allow movement, either having been stretched or cut or assisted by elastic traction. A determined patient is needed, able to co-operate in the face of pain; and there must be an extended wrist to give full power to flexion by flexor tendons free of scar and in continuity, and extensor tendons, too, free of adhesions, to allow flexion to occur.

MISCELLANEOUS

In two cases of totally disabled hands from crush injuries which had resulted in metacarpal fracture with flexor and extensor tendon fixation to deep and skin scar, and with wrist drop, MP extension and fixation, and thumb adduction, a considerable degree of recovery was possible by the following measures: (i) Freeing tendons, (ii) resecting portions of metacarpals where scar and deformity made recurrence of adhesions to tendons inevitable, (iii) flexing MP joints, (iv) fixation by plaster of Paris with wrist extension, MP joint flexion and crossbar leverage for fingers, (v) elastic traction, and (vi) electric stimulation of flexors and extensors at an early stage.

Tendon Transplants and Grafts

In late poliomyelitis cases, the results of tendon transplants have been very satisfactory, combined sometimes with arthrodesis of the wrist.

In spastic cases, operations may relieve deformity, but rarely give much improvement in function.

Skin Coverage

Perhaps the most important advance in hand surgery in recent years is the use of skin coverage when needed. Time and again early skin placement over tendons, bone and exposed joints has saved limb and function.

One can wait a week to assess the degree of necrosis in skin and tendon, etc., for it is not always possible immediately to assess the degree of damage, which is almost always more than the first impression suggests. Even a longer lapse of time does not prevent coverage. When one is in doubt in the early stages, a dressing of tulle gras tied over with some pressure will almost always ensure a healthy surface on inspection at the end of a week.

In a case seen 18 days after gross injury, in which the dorsal structures of the right forearm, wrist and dorsum of the hand were lost, bone denuded of periosteum was visible, including lengths of radius, ulna and metacarpals. Free pus escaped from the wrist area, and the ring and small fingers and the metacarpals were loose and necrotic.

Carpal bones were excised, and the flexor tendons could be seen from the dorsum bathed in pus. Free drainage was established in the palm, and a dressing of tulle gras was sewn into place with pressure.

A week later, a large abdominal flap was used as coverage. The exposed bones were nibbled until bleeding occurred. Healing took place without sinuses and, within a short time, extensor tendon grafts to the abductors and extensors of the thumb and remaining fingers could be done through the flaps.

One learns from this case that, even after 18 days of exposure of bone and tendons, coverage is still possible, and this has been our experience with other cases of exposed bones and joints in neglected compound fractures and dislocations of the ankle and tibia.

Different methods of reconstructing thumbs can be accomplished, such as by skin pedicle from the abdomen, and later bone grafting. Transplantation of a stump of an amputated middle finger and metacarpal to the thumb has been used successfully, and so has rotation of a metacarpal. Each case should be considered on its own merits; we are always assisted by a good patient, and the natural adaptability of remaining digits for practical use.

SUMMARY

In this talk, an effort has been made to explain the anatomical reasons for recovery of a correctly positioned hand as against the deterioration to a fixed claw hand in a poorly positioned hand.

The deformity of hyperextension of proximal interphalangeal joints with flexion of distal interphalangeal joints has been shown to be due to several interesting mechanical causes.

The concept of alternate immobilization to retain and restore finger movements is propounded. Early skin coverage by flaps of exposed tendons, bone and joints is strongly advocated at the moment the necrosis of covering skin is discernible.

IRON DEFICIENCY ANAEMIA IN BANTU INFANTS, AND ITS ASSOCIATION WITH KWASHIORKOR

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South African Institute for Medical Research, and Department of Paediatrics, Baragwanath Hospital, Johannesburg

Other than a reference made in 1948 by Altmann and Murray[1] to 4 cases of anaemia in kwashiorkor with a microcytic blood picture, iron-deficiency anaemia in Bantu children in South Africa has received very little attention in the literature. It has been suggested[2] that this form of anaemia is rare in the South African Bantu on account of the apparently high intake of iron, derived from iron utensils used in the preparation of their food. At Baragwanath Hospital, Johannesburg, however, severe iron-deficiency anaemia is encountered in Bantu children, and it is the purpose of this communication to draw attention to its occurrence in this group, to stress the relationship to kwashiorkor, and to report the favourable response of the anaemia to therapy with intramuscular iron.

DIAGNOSTIC CRITERIA

In the series of cases here reported, iron-deficiency anaemia was diagnosed when the haemoglobin value was less than

10 g.%, the mean corpuscular haemoglobin concentration (MCHC) was 30% or lower, and hypochromic erythrocytes were present in the blood. Children suffering from chronic infection or blood loss were excluded from the study. The distinction between iron-deficiency anaemia and the hypochromic anaemia which may accompany chronic infection was made largely on clinical grounds, since the differentiation of sideropenic and non-sideropenic forms of hypochromic anaemia is not easy in infants, because changes cannot be detected in iron stores in the bone marrow. Infection was often the reason for admission to hospital of children with iron-deficiency anaemia, but only cases where infection was of but a few days' duration were included in the study. Patients in whom iron deficiency was associated with megaloblastic anaemia were excluded.

RESULTS

The features of 60 cases occurring in children admitted to the paediatric wards fulfilling the above diagnostic criteria are presented.

1. *Age.* The ages of the infants and children ranged from 3 months to 3½ years, but 82% of cases occurred between 6 and 18 months of age.

2. *Seasonal incidence.* Cases occurred at all times during the year, with the distribution as follows: 4 in January, 1 in February, 7 in March, 4 in April, 4 in May, 6 in June, 8 in July, 2 in August, 4 in September, 8 in October, 9 in November and 3 in December.

3. *Perinatal factors.* Six infants (10%) had been premature. In none of the cases was there a history of haemolytic or haemorrhagic disease of the newborn or of cord haemorrhage.

4. *Feeding history and the iron content of the feeds.* Of the 60 cases, 4 were breast-fed at the time of admission to hospital and were receiving complements of meat, vegetables, porridge and milk; 46 were fed on maize porridge and water, with occasional supplements of milk, vegetables and meat; and 10 received maize porridge only. In order to assess the frequency of the use of iron pots in the preparation of the infants' feeds by the local Bantu population, 300 mothers attending the out-patient department were questioned. None of these used iron pots, all the cooking utensils employed being either enamel or aluminium, but 10% admitted to the use of iron tins for the souring of porridge after cooking. The iron content of some of the maize mixtures on which malnourished children were fed (chosen at random) has been determined (Table I). The total daily iron intake varied between 2·1 and 4·5 mg. (mean 2·9 mg.).

TABLE I. IRON CONTENT OF FEEDS GIVEN TO BANTU INFANTS AND CHILDREN

Age (mths.)	Type of Feed	Feed Analysed: Volume (ml.)	Feed Analysed: Dry weight (g.)	Feed Analysed: Iron Content (mg.% of dry weight)	Volume of Feed per Day (oz.)	Iron Intake per Day (mg.)
21	Maize meal and water	220	13·77	2·1	22	2·5
18	Maize meal and water	210	25·44	4·0	27	4·5
15	Maize meal and water	180	17·73	3·0	30	2·7
12	Maize meal and dilute milk	113	19·70	2·1	25	2·8
9	Maize meal and dilute milk	190	27·57	2·3	25	2·1

5. *Features of malnutrition.* Only 6 of the 60 children investigated were in a reasonable state of nutrition. The remaining 54 were malnourished, all showing depigmentation of hair; acute nutritional dermatosis was present in 20, 24 were oedematous, and 17 were markedly wasted. All were under-weight for age.

6. *Admission diagnosis.* In 12 infants (20%) anaemia was the diagnosis on admission. Many children were admitted with more than one diagnosis. The other diagnoses were as follows: Malnutrition 33, gastro-enteritis 25, pneumonia 15, rickets 5, whooping cough 3, heart failure 2, congenital heart disease 1, glandular fever 1, and acute arthritis 1.

7. *Peripheral blood.* The haemoglobin value ranged from 3·6 to 9·9 g.% (mean 8·13 g.%) and the MCHC from 22 to 30% (mean 28·6%). The deficiency in haemoglobin often did not parallel that in the MCHC or the degree of hypochromia of the red cells in the smears. With haemoglobin values greater than 9·0 g.% the red cells often exhibited marked hypochromia. The degree of anisopoikilocytosis was usually moderate, with cigar-shaped poikilocytes often prominent. A slightly raised reticulocyte count was a common finding, the counts on admission ranging from 1 to 8%. Platelets were present on the smears in normal number. The leucocyte picture frequently reflected the condition which had brought the child to hospital.

8. *Bone marrow.* Puncture of the tibial or iliac crest was carried out in 17 random cases. The marrow specimens were generally very cellular, and an erythroid reaction was noted in all cases where the myeloid-erythroid ratio was determined; the ratio was less than 1·5:1 in all. The polychromatic and pyknotic normoblasts showed evidence of iron deficiency; the degree generally paralleled that of the anaemia and hypochromia of the red cells, and was often very conspicuous. The normoblasts were small and deformed, and contained very little cytoplasm. The myeloid series was usually normal.

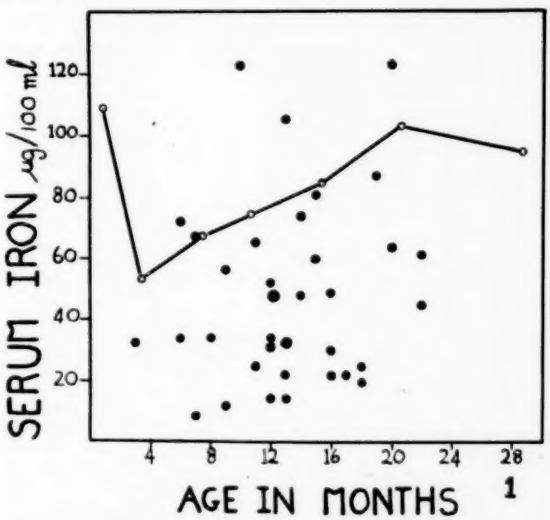

Fig. 1. Serum-iron values in 35 children. 0——0 refers to the average normal values in infancy and childhood according to Sturgeon (1954).

9. *Serum iron.* Serum iron was determined in 35 random cases, before any iron therapy. These values are shown (Fig. 1) against the average normal values during infancy according to Sturgeon.[3] In 10 infants serum iron was 30 μg. per 100 ml. or less.

10. *Treatment*

The cases were divided into 3 groups, according to the treatment.

(*a*) *No specific haematinics:* 19 infants with anaemia, usually of moderate degree, received no iron therapy. Rise in haemoglobin value on ward diet occurred in less than 50% of these cases. The reticulocyte response was never greater than 5% and a rise in haemoglobin was usually not accompanied by rise in MCHC or diminution in the degree of hypochromia of the erythrocytes in the blood smears.

(*b*) *Oral iron:* 13 infants were treated with a colloidal iron preparation in a dose of 90 mg. metallic iron daily. We could not assess the response in 3 of these patients because 2 of them were given blood transfusions and 1 had intramuscular iron added early in the course of therapy. Of the remaining 10 patients treated, only 4 showed a significant rise in haemoglobin (Fig. 3); one case showed a fall in haemoglobin from 8·2 to 6·5 g.%. In one case the haemoglobin level rose from 8·1 to 8·4 g.% after 30 days' treatment with oral iron, and when this was changed to intramuscular iron therapy the haemoglobin level rose to 10·4 g.%, 10 days after the first injection.

Seven of the 10 cases in whom haemoglobin responses were assessed had reticulocyte responses assessed concurrently (Fig. 2). The maximum observed reticulocyte response was only 9% after 15 days of therapy with oral iron.

(*c*) *Intramuscular iron:* 25 infants were treated with an intramuscular iron-dextran* preparation. The dosage was calculated on the initial haemoglobin level, the total amount administered varying between 150 and 450 mg. The effect of therapy could be satisfactorily assessed in 15 cases. All 15 responded as judged by a rise in haemoglobin of at least 2 g.% or reticulocyte response of greater than 10%, or both. The reticulocyte responses in the cases treated with intramuscular iron are contrasted in Fig. 2 with those who received oral iron, and the haemoglobin increments in Fig. 3. The reticulocyte response on intramuscular iron was often very marked, and was usually apparent by the 3rd or 4th day after the first injection, the maximum count being reached on the 5th or 6th day. In only 1 infant, where the initial haemoglobin value was 8·8 g. per 100 ml., was reticulocytosis after therapy not observed. The results of treatment with intramuscular iron are often remarkable. In 2 infants with initial haemoglobin values of 3·6 and 4·4 g.% the haemoglobin had risen to 9·9 and 10·8 g.% respectively by the 15th day of therapy. (Of the other 10 cases, one, a child admitted with acute liver necrosis, died on the 4th day of therapy, 1 child received a blood transfusion as well as iron, and in 8 follow-up was inadequate.)

* Imferon, Bengers Laboratories.

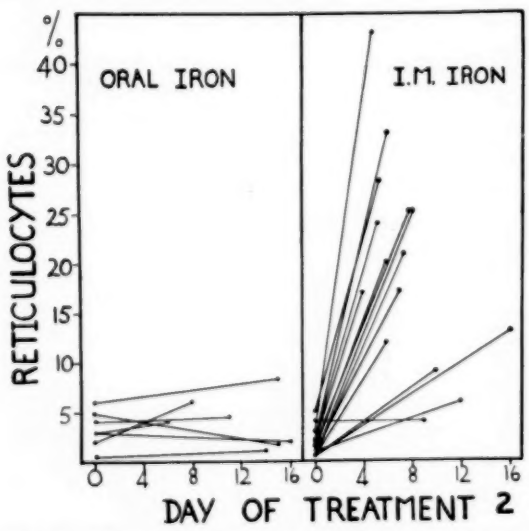

Fig. 2. Reticulocyte response to oral iron, and to intramuscular iron.

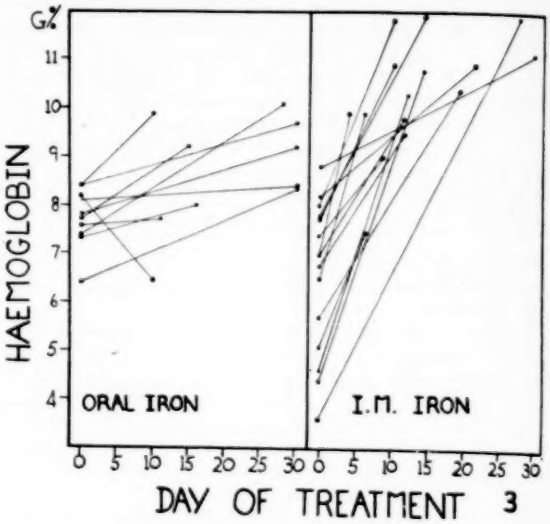

Fig. 3. Haemoglobin increment after treatment with oral iron, and intramuscular iron.

DISCUSSION

The exact role of a number of aetiological factors in the pathogenesis of iron-deficiency anaemia in infancy remains undecided. In the premature infant, rapidity of growth associated with a defective prenatal iron store (small red-cell mass) leads to a high incidence of iron-deficiency anaemia. Prematurity does not appear to be of much direct significance in the aetiology of the present group of cases, only 5% of them being premature. Diminished iron stores at birth is probably not a significant factor in our cases. The infant's iron stores reside mainly in his haemoglobin, and haemoglobin concentration at birth does not differ significantly in the 2 groups. (Although the birth weight of the newborn Bantu may be slightly less than that of the White population, the difference is not significant.) Other possible aetiological factors, such as jaundice of the newborn and haemorrhage

from the cord or elsewhere, do not play any part. There is no obvious seasonal incidence.

Nutritional factors, however, are of prime importance. Only half the infants were breast fed for as long as 3 months. The foods onto which the children had been weaned, with only an occasional exception, contained no adequate source of iron. The results of the determination of the iron content of the maize feeding mixtures employed (Table I) indicates that the iron intake of the Bantu infant with kwashiorkor falls short of the recommended dietary allowance. Iron cooking utensils are not in use in this urban Bantu population. Even assuming that normal absorption of 10% of the dietary iron occurs, the daily iron assimilated is deficient. In addition, absorption of iron in malnourished infants is probably poor. Low vitamin-C intake[4] and the frequent occurrence of hypochlorhydria[5] and of diarrhoea, probably all result in poor absorption of oral iron. The main cause of the iron-deficiency anaemia accompanying kwashiorkor would thus appear to be deficient iron intake, possibly aggravated by poor absorption.

The possibility of poor absorption of iron in kwashiorkor may explain why the treatment of iron-deficiency anaemia with oral iron in these cases has been found to be unsatisfactory. The results of oral iron therapy were unpredictable and were far inferior to those obtained with intramuscular iron. Moreover, oral iron may aggravate the gastro-intestinal disturbance commonly present in kwashiorkor. Ward diet without additional iron therapy did produce a rise in the haemoglobin in some cases, but not in the MCHC, and the degree of hypochromia did not diminish.

Intramuscular iron was found to be effective in 11 out of 12 cases of kwashiorkor reported by Trowell and Simpkiss[6] from Uganda, and the present study confirms the satisfactory results obtained with this preparation. Kwashiorkor as seen in South Africa as opposed to Central Africa is not complicated by helminthic infestation or malaria. In the present series no case was encountered where iron deficiency anaemia did not respond to treatment with intramuscular iron. With the intramuscular route it is possible to provide the child with iron stores, a factor of considerable significance in that the child with kwashiorkor, on discharge from hospital, may return to his previous poor dietary pattern.

CONCLUSIONS AND SUMMARY

The clinical and haematological findings and response to treatment in 60 Bantu children with iron deficiency anaemia are presented.

Of these 60 cases 54 showed the features of kwashiorkor.

The maize mixtures almost universally employed in the feeding of Bantu infants are often deficient in iron.

The serum-iron content, determined in 35 children, was generally low.

The results of the treatment of the anaemia with oral iron are unpredictable. The response to treatment with intramuscular iron was highly satisfactory in all the cases in which treatment could be assessed. Therapy with intramuscular iron is suggested as the treatment of choice of the iron-deficiency anaemia investigated.

We wish to thank Drs. D. A. Sutton and N. J. van Rensburg for the determination of the iron content of the feeding mixtures, and Drs. E. Kahn, S. Wayburne, H. B. W. Greig and R. Cassel for their advice. We also wish to thank the Director, South African Institute for Medical Research, for facilities to perform this study, and the Superintendent of Baragwanath Hospital, for permission to publish this paper.

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CEREBRAL PALSY*

BEN EPSTEIN, *Chairman of the Board of Management, Pretoria School for Cerebral Palsy*

Today's function is another milestone in the history, not only of the Pretoria School for Cerebral Palsy, but also of the whole cerebral palsy movement in South Africa. The school was started in 1950 with a grant of £100 from the Cripple Care Association, and a great deal of enthusiasm and faith. However, even the most enthusiastic and hopeful member of the original committee could not have foreseen that within the short period of nine years we would be witnessing the erection of this magnificent building, designed so thoughtfully and carefully for the cerebral palsied child.

This result has not been achieved without great effort on the part of many people. We must acknowledge our indebtedness to the State that has subsidized the running costs of the school and contributed the sum of £80,000 towards the capital expenditure on the buildings. It would not have been possible for us to proceed without this aid. We must thank sympathetic officials in the Department of Education, Arts and Science, from Dr. C. M. van Antwerp, whom we are happy to have here on this platform as our guest speaker, to the Secretary, Mr. J. J. P. Op 't Hof. We also acknowledge the help and interest of the late Minister, the Hon. J. H. Viljoen, whose passing is so much regretted. We could not have had a kinder Minister at the time we started our work.

What does this school signify? What does it represent? The bricks and mortar, even the aesthetic beauty of the building, are insignificant in relation to the work that will be done inside the walls. We have paid some attention to the need for an architecturally sound building, well suited to the natural environment, but our greatest efforts have been devoted to the design of a building where handicapped children could learn, and devoted staff could teach. This result we think we have achieved, thanks to our architects, Messrs. Todd, van Schaik, and Austin, working in cooperation with Dr. Calderwood and other experts from the National Building Research Institute.

This building, perhaps, has the greatest significance for parents. A family with a handicapped child is a handicapped family. When parents are told that nothing can be done for a handicapped child, the bottom falls out of their world; and this may happen to young parents with their first child, to parents of an only child, and to parents of many children. The handicapped child always limits their horizon, and forces limitations on the whole family. Only the hard and callous could fail to sympathize with them in their agony and in the feelings of helplessness and hopelessness which they must endure. Such helplessness was the lot of the parents of cerebral palsied children in Pretoria until 1950, when the school was opened with only 4 children. The shadow has been lifted, and the burden diminished. The reponsibility is now being shared by fellow-parents, and by a large organization—the National Council for the Care of Cripples in South Africa. Hope and courage have been instilled.

* Chairman's address at the laying of the foundation stone, Pretoria School for Cerebral Palsy, 24 June 1959.

The parents have been wonderful. What you see today is primarily their work. Tribute is being paid to the parents through the person of Mrs. P. Stals Robertson, Chairman of the Parents' Association, who has dedicated her life to the crippled. I have no doubt, whatsoever, that without Mrs. Robertson's initiative and self-sacrifice this school would not have had the distinction of being the first school in South Africa specifically designed for the teaching and treatment of the cerebral palsied child.

For the community of Pretoria this school is the outward symbol of the fact that it accepts responsibility for its handicapped people. The standard of civilization of a community is reflected in the services it is ready to render to its handicapped members: its physically and mentally handicapped, and its under-privileged people of whatever race, colour, or creed. I look forward to the day when the word 'charity' will have lost its sting, and when necessitous individuals will, as a right, have a claim to the resources of the country.

It is economically important for a country that every person who is in any way capable of doing productive work should make use of his abilities. It is unsatisfactory to allow large numbers of people to draw invalidity grants when some form of sheltered employment can be arranged for them. This school shows the faith of the Pretoria community in this principle, and the faith of the Department under which we function.

This school represents for me the culmination, although, I hope, not the conclusion of many years of interesting and satisfying work. Medical practice gives opportunity for service which few professions can give. I feel that I am fortunate, indeed, to be a member of this profession. I am very conscious of the great honour that has been conferred on me by the Board of Management in inviting me to lay this foundation stone. I accept the honour with humility, in the realization of my limitations in the face of the multiple problems of cerebral palsy. It is a great privilege to be able to render a service in a field that has, until recently, been completely neglected.

THE MEDICAL ASSOCIATION OF SOUTH AFRICA

ANNUAL REPORT OF THE CHAIRMAN OF FEDERAL COUNCIL FOR THE YEAR ENDED 30 JUNE 1959

Obituary. It is with deep regret that we record the loss through death of the following members: Drs. L. Beukes, E. M. Chubb, J. Conroy, A. F. W. Davis, J. S. du Toit, G. M. Fox, T. A. Fuller, A. G. H. Hay-Michel, W. B. Hudson, L. N. Kaplan, H. G. Lee, Jack A. Levitt, T. Lotter, P. J. Mentz, P. J. Olivier, D. D. Palmer, R. L. Paterson, N. B. Peacock, D. H. Pfeiffer, J. A. Willenge Prins, J. C. Rabie, R. Robins-Brown, R. G. Simons, W. A. Twigg, G. V. Vlotman, E. H. Walker, H. Wallace-Jones, P. S. Wallach, H. W. Wier and J. Weinberg.

Membership. During the past year there has been an overall decrease in members of 26, the total membership now being 5,502. In addition there are 76 student members. Members are distributed as follows: Border Branch 216, Cape Eastern Branch 57, Cape Midlands Branch 229, Cape Western Branch 1,220, East Rand Branch 266, Griqualand West Branch 89, Natal Coastal Branch 557, Natal Inland Branch 193, Northern Transvaal Branch 532, O.F.S. and Basutoland Branch 382, Southern Transvaal Branch 1,220, South West Africa 79, Transkei Branch 79, Vaal River Branch 68, unattached members 278, Emeritus members 29, Honorary members 8. It was anticipated that there might be some resignations as a result of increasing the Association's annual subscription, but these were fewer than was expected.

Honours. During the year under review the Federal Council agreed to the award of the Association's Gold Medal for distinguished service to the Association and the medical profession to Dr. T. Shadick Higgins, one time Medical Officer of Health of Cape Town and later Editor of the *South African Medical Journal*. Bronze Medals for meritorious service to the Association for 1959 were awarded to Dr. Lewis S. Robertson (Southern Transvaal Branch), Dr. C. Seymour Heymann (Southern Transvaal Branch), Dr. P. V. H. Wagner (Border Branch) and Dr. J. N. W. Loubser (O.F.S. and Basutoland Branch). In addition Council honoured Dr. A. Temple Thurston, Dr. L. R. Smuts, Dr. I. P. Schabort and Dr. J. M. Watt by electing them to Emeritus Membership.

Annual General Meeting. The Annual General Meeting of the Association for the year 1958 took place in Pretoria on 1 October 1958. At the conclusion of the formal business Dr. R. Schaffer of Queenstown was inducted as President by Dr. H. Grant-Whyte, the retiring President. The meeting was then adjourned until the evening, when Dr. Schaffer delivered his Presidential Address and awards were presented. The adjourned Annual General Meeting was followed by a reception.

Congress. No Congress was held during the year under review but preparations have been made for the holding of a Congress in East London in September 1959.

The following were some of the Group Congresses held during the year under review: The 2nd Congress of the Urological Association of South Africa, Cape Town, 17-19 July 1958; the 7th Interim Congress of the South African Society of Obstetricians and Gynaecologists, Pretoria, 4-6 August 1958; the 3rd Congress of the South African Paediatric Association, Pretoria, 9-11 October 1958; the 1st Scientific Congress of the Association of Physicians of South Africa, Cape Town, 8-10 January 1959; the 8th Interim Congress of the South African Society of Obstetricians and Gynaecologists, Bloemfontein, 2-5 March 1959; and the National Meeting of the Dermatological Sub-group of the Dermatology and Venereology Group, Johannesburg, 28-29 March 1959.

Federal Council. There have been 2 meetings of the Council. The first of these was held in Pretoria on 1-3 October 1958 and the second in Johannesburg on 8-10 April 1959. The average attendance at these meetings was 51 out of a total membership of 60. The Executive Committee has met on 5 occasions. Two meetings were held on the day preceding the 2 Council meetings and 3 special meetings have been held, in Pretoria in August 1958, in Cape Town in February 1959 and in Pretoria in May 1959.

Committees of Council

The Head Office and Journal Committee continues to play its part in the administrative and financial affairs of the Association and its *Journals*. Dr. A. P. Blignault was appointed Editor as from 1 January 1959 after being Assistant-Editor for some 7 months, while the retiring Editor, Dr. T. Shadick Higgins, has remained on to help as acting Assistant Editor. There has been re-organization in the Accounts Department which has led to the retrenchment of certain clerks.

The Federal Ethical Committee met on one occasion during the year and dealt with certain other matters by correspondence.

The Central Committee for Contract Practice has continued to do a considerable amount of routine work and has dealt with a number of new applications for recognition. Dr. L. O. Vercueil, who has been Chairman of the Committee for a number of years, has resigned from this position although he has agreed to continue to serve as a member of the Committee. Council has decided that the work of this committee be undertaken at the Pretoria office and that Dr. Marchand, Associate Secretary, be transferred in order to carry on with this important task.

The Parliamentary Committee has had frequent meetings and has dealt with a considerable amount of business, most of which has been successfully concluded with advantage to members of the Association.

The Workmen's Compensation Act Sub-committee has continued to act in liaison with the Commissioner and has attended to a number of routine matters.

Medical Services Plan. In former years progress had been reported under the Sub-Committee on the Economics of Medical Practice which was discharged in April 1958. It was then reported that the Medical Association had agreed to sponsor the Medical Services Plan and the Southern Transvaal Branch undertook to watch the interests of the Association as regards its sponsorship. The draft constitution was published in the *Journal* for 18 April

(33, 337) for general information and the Plan was put into operation on 1 June 1959.

Journals. Reference has already been made to the appointment of Dr. Blignault as Editor. The cover of the weekly *Journal* was altered to its present form from 1 January 1959 and strict control over the text pages should lead to a better financial position at the end of the year. The supply and quality of the articles published is as high as it has ever been. The quarterly *South African Journal of Laboratory and Clinical Medicine* continues to be published and fulfils a definite function in providing a vehicle for the more specialized article.

Branches, Divisions and Groups continue to hold regular meetings and to serve the members resident in their areas. Although it is essential that business should receive attention, it is the clinical meetings that are most appreciated by the average member.

World Medical Association. The Twelfth General Assembly was held in Copenhagen on 15-20 August 1958. Dr. Lewis T. Robertson, of Johannesburg, represented our Association on that occasion.

Finance. After a series of difficult years during which considerable inroads have been made into the capital of the Association the year 1958 ended with a deficit of £8,805. The accumulated funds now stand at £13,348, most of which consists of fixed property or unrealizable assets. The raising of the subscription, which was overdue in a world of changing values, will make it easier to carry on the work of the Association and a small surplus has been estimated for the end of 1959. At this rate it will take many years to build up the capital which has been lost. It must be obvious to all that if the Association is to maintain the standard of its service to members, the service must be paid for by the members' subscriptions which must be adequate for the purpose.

Benevolent Fund. There have been 31 beneficiaries of this Fund during the year, one of whom died in May 1959. The accumulated funds stood at £47,549 on 31 December 1958 and during the year grants totalling £3,746 9s. 4d. were made.

Library Grants. Grants have been made to the libraries of the five medical schools totalling £800 during the year under review.

Medical Agencies. The agencies maintained in Cape Town and Johannesburg continue to render valuable service to members, who are reminded that they were established to be of assistance to all. The Medical Insurance Agency continues also to provide a service to members through various forms of insurance—public liability, motor-car, life and endowment—and the many forms of general insurance cover.

Conclusion

On behalf of the Council I would thank all who have contributed to the work of the Association. I refer particularly to the honorary officials and members of the various committees of Council and of the Branches, Divisions and Groups. Many of these officials have made great sacrifices of time and leisure in the interests of the Association and of their colleagues in the profession.

I would also extend the thanks of the Council to the members of the staff, both medical and lay, who have served the Association well.

J. H. Struthers
Chairman of Council

Pretoria
3 July 1959.

PROGRAMME: 42ND MEDICAL CONGRESS (M.A.S.A.), EAST LONDON, 27 SEPTEMBER–3 OCTOBER 1959

Plenary Sessions

Tuesday a.m., 29 *September*

CANCER

Recent advances in cancer therapy with special emphasis on radiation therapy: Prof. R. McWhirter (UK).

Experimental cancer and environmental factors in the aetiology of cancer: Dr. H. Stewart (USA).

The demography of cancer in South Africa: Prof. E. H. Cluver.

The problems of cancer survey in a primitive area: Dr. R. J. W. Burrell.

Factors in cancer demography in the Cape Division—a preliminary report: Dr. J. Muir Grieve.

A cancer survey in Lourenço Marques: Dr. M. Prates.

Wednesday a.m., 30 *September*

HEART DISEASE

(*a*) *Coronary disease.* The aetiology and diagnosis of ischaemic heart disease: Prof. G. Burch (USA).

The treatment of coronary heart disease: Dr. M. M. Suzman.

(*b*) *Cardiac surgery.* Cardiac surgery: Sir Russell Brock (UK).

Repeat mitral valvotomy: Mr. L. Fatti.

Thursday a.m., 1 *October*

TUBERCULOSIS—YESTERDAY, TODAY AND TOMORROW

Basic principles in the epidemiology and control of tuberculosis: Dr. B. A. Dormer.

The research aspects of tuberculosis in South Africans: Dr. B. Sampson.

The control of tuberculosis in urban areas: Dr. E. A. MacIldowie.

The control of tuberculosis in rural areas: Dr. J. F. Taute.

Problems of tuberculosis as seen by an orthopaedic surgeon: Mr. R. Percy-Lancaster.

Sectional Meetings

ANAESTHETICS

Monday 28 *September*

Cardiopulmonary bypass in cardiac surgery: Dr. J. Ozinsky.

The experimental use of fluothane anaesthesia in open-heart surgery with cardiopulmonary bypass: Drs. A. B. Bull, G. J. Rossouw, Prof. J. Kench and Dr. C. N. Barnard.

Open-heart surgery using the disc oxygenator and roller type pumps: Mr. L. A. du Plessis.

Film: Preparation and operation of the disc type of oxygenator: Mr. L. A. du Plessis.

Tuesday 29 *September*

Unusual accidents and incidents of anaesthesia: Dr. J. N. Abelsohn.

Temperature changes in children during general anaesthesia: Drs. G. G. Harrison, A. B. Bull and H. J. Schmidt.

Halothane (fluothane) in anaesthetic practice: Dr. J. T. Russell.

Friday 2 *October*

The treatment of tetanus neonatorum: Dr. R. Wright (with paediatrics and otorhinolaryngology).

Cardiac arrest—a review of 300 cases: Drs. O. V. S. Kok and C. Kitay (with film).

Film: The treatment of cardiac arrest: Sir Russell Brock (UK).

Blood loss in paediatric surgery: Dr. J. A. Pretorius.

Porphyria and anaesthesia: Drs. R. A. M. Dyke and G. Dean.

GENERAL PRACTICE

Monday 28 *September*

The Zulu diabetic: Dr. G. D. Campbell.

Some social and familial aspects of ischaemic heart disease: Dr. B. Kaplan.

Urticaria—an aetiological and therapeutic problem: Dr. D. Ordman.

Manipulative orthodysarthrics: Dr. P. H. Dalgleish.

Tuesday 29 *September*

Hypertension in pregnancy: Dr. J. M. Samson.

The value of hypotensive drugs in the treatment of eclampsia: Dr. M. G. H. Mayat.

Treatment of acne: Dr. R. Ravin (USA).

Wednesday 30 *September*

Medical hypnosis in general practice: Dr. A. N. Sacks.

Hypertension with unilateral kidney disease: Drs. A. J. Tinker and M. B. M. Denny.

Further observations in the radiological investigation of peripheral vascular disease: Dr. M. B. M. Denny.

Premenstrual tension syndrome: Dr. F. Benjamin.

Friday 2 *October*

The psychiatric approach to tuberculosis: Dr. B. W. Crowhurst Archer.
Congestive cardiac failure: Prof. G. Burch (USA).
Cardiac neurosis: Dr. R. Schaffer.
Radiotherapy of the common primary malignant conditions of the skin: Dr. M. Weinbren.
The changing status of amoebiasis: Dr. R. Elsdon-Dew.

HOSPITAL ADMINISTRATION

Monday 28 *September*

The King Edward VIII Hospital central sterile supply department: Dr. S. Disler.
Artificial insemination: group discussion.
Termination of pregnancy: group discussion.

Tuesday 29 *September*

The administration of blood transfusion services in hospitals: group discussion.
Mediese superintendente of leke administrateurs vir algemene hospitale: Dr. J. de Beer.
Donor banks—blood, bone, cornea: group discussion.

Wednesday 30 *September*

Surgical deaths: group discussion.
Sudden deaths: group discussion.
Losses in hospitals: group discussion.

Friday 2 *October*

All-day outing being arranged by the secretary of the Section.

MEDICINE

Monday 28 *September*

Atrial septal defect: Drs. M. M. Zion, J. L. Braudo and B. A. Bradlow.
Variations in the atrial sound: Dr. J. B. Barlow.
The conception of rheoplethysmography with selected applications: Prof. G. Burch (USA).
Lung cancer among White South Africans: Dr. G. Dean.

Tuesday 29 *September*

Experience with the artificial kidney: Dr. M. L. Simenhoff and Prof. L. Eales.
The leukaemias: Dr. M. M. Suzman.
Studies of venous tone in intact man: Prof. G. Burch (USA).

Wednesday 30 *September*

Hypertension with unilateral kidney disease: Drs. A. J. Tinker and M. B. M. Denny.
The value of amyl nitrite in the diagnosis of systolic murmurs: Drs. L. Vogelpoel, M. Nellen, A. Swanepoel and V. Schrire.
A study of the electrocardiogram in transposition of the great vessels: Dr. T. C. Meyer.
New concepts in sexual differentiation: Drs. R. Hoffenberg and W. P. U. Jackson.

Friday 2 *October*

The cutaneous manifestations of porphyria: Prof. L. Eales.
Endocrine function in malnutrition: Drs. A. O. Lurie, C. P. Dancaster and W. P. U. Jackson.
The use of iodine ^{131} in the investigation of thyroid disease: Dr. R. Hoffenberg and Elizabeth Black, B.Sc.
Congenital aortic stenosis: Drs. J. L. Braudo and M. M. Zion.
New clinical applications of spatial vectorcardiography: Prof. G. Burch (USA).
Phonocardiography before and after open-heart surgery: Drs. M. Nellen, L. Vogelpoel, V. Schrire, A. Swanepoel and C. N. Barnard.
Approach to prediction of diabetes: Dr. W. P. U. Jackson.
Acyanotic Fallot's tetralogy: Drs. M. M. Zion, J. L. Braudo and S. C. Heymann.
The medical and surgical aspects of arterial thrombo-embolism in rheumatic heart disease: Prof. A. Ravin (USA) and Dr. J. Grow.
Porphyria and anaesthesia: Drs. R. A. M. Dyke and G. Dean.
The changing status of amoebiasis: Dr. R. Elsdon-Dew.

NEUROLOGY, PSYCHIATRY AND NEUROSURGERY

Monday 28 *September*

Pyogenic infections of the nervous system and its coverings: Prof. J. F. P. Erasmus.

Tuesday 29 *September*

The prevention of mental illness and an analysis of the causes of delinquency: Dr. H. M. Wolfsohn.
A case of psychosexual sterility: Dr. F. B. Proksch.

Wednesday 30 *September*

The treatment of congenital hydrocephalus: Dr. M. J. Joubert.
Film: Pudenz operation for hydrocephalus: Dr. M. J. Joubert.
Radical surgical treatment of cranio-pharyngiomas and follow-up studies: Mr. E. M. Kerr.

Friday 2 *October*

Medical hypnosis in general practice—a practical demonstration: Dr. A. N. Sacks.
Psychiatric approach to tuberculosis: Dr. B. W. Crowhurst Archer.
A neurological pellagrinous syndrome occurring in non-Europeans on the Witwatersrand: Dr. S. Jacobson.

OBSTETRICS AND GYNAECOLOGY

Monday 28 *September*

Cancer *in situ* of the cervix: Prof. T. Antoine (Vienna).
Some pre-operative considerations in cervical carcinoma: Prof. E. D. Crichton.
A review of eight years' work (1951-1958) on cancer of the cervix uteri: Prof. J. T. Louw.
Methods for early detection of cervical carcinoma: Prof. T. Antoine (Vienna).

Tuesday 29 *September*

Hypertension in pregnancy: Dr. J. M. Samson.
The value of hypotensive drugs in the treatment of eclampsia: Dr. M. G. H. Mayat.
Observations on the value of cephalo-pelvimetry undertaken during trial labour: Prof. E. D. Crichton.

Wednesday 30 *September*

Urinary infection associated with catheterization in gynaecology and obstetrics: Dr. A. Viljoen.
Obstetric deaths in hospital practice: Dr. D. K. Quinlan.
The possibilities of determining the ovulation time: Prof. T. Antoine (Vienna).
Premenstrual tension syndrome: Dr. F. Benjamin.

Friday 2 *October*

Hormonal response of endometriosis: Prof. T. Antoine (Vienna).
Facilitation of labour by abdominal decompression: Prof. O. S. Heyns.
Film: Wertheim's hysterectomy: Prof. J. T. Louw.
Film: Basset's operation: Prof. F. G. Geldenhuys.
Some observations relating to pregnancies following Caesarean section among Africans in Natal: Dr. A. H. Lasbrey.
Cardiac arrest: A review of 300 cases: Drs. O. V. S. Kok and C. Kitay.
Blood loss in paediatric surgery: Dr. J. A. Pretorius.

OPHTHALMOLOGY

Monday 28 *September*

History of trachoma in South Africa: Dr. R. St. H. Warren.
Prevention and cure of trachoma: Drs. J. G. Scott and I. Taylor.
The result of first field trial in treatment of trachoma: Dr. J. G. Scott.
Film: Trachoma in South Africa: Dr. J. G. Scott.

Tuesday 29 *September*

Statistical evaluation of controlled field trial in trachoma: Dr. A. M. Adelstein.
The growth and characteristics of the virus of trachoma: E. Whitney and Dr. J. H. S. Gear.

Wednesday 30 *September*

Present-day ophthalmological treatment in Europe particularly in the use of light coagulation: Dr. W. J. Levy.
Group discussions.

Friday 2 *October*

Group discussions to be arranged by the secretary of the Section.

ORTHOPAEDICS

Monday 28 *September*

The diagnosis and treatment of low back pain—practical demonstration: Mr. R. L. Diveley (USA).

Operative treatment of prolapsed lumbar discs: Messrs. G. T. du Toit and D. Roux.

Some pitfalls of disc surgery: Mr. L. Mirkin.

Cervical disc syndrome: Mr. R. C. J. Hill.

Tuesday 29 *September*

Fractures of the carpal scaphoid: Mr. R. L. Diveley (USA).

Reduction of severity and frequency of injury in car accidents: Mr. G. T. du Toit.

Diametric fractures of the pelvis: Mr. G. F. Dommisse.

Wednesday 30 *September*

Clinical demonstration—Orthopaedic department Frere Hospital, East London.

Friday 2 *October*

Tibialis posterior transfer in congenital club foot: Mr. M. Singer.

Treatment of the calcaneous foot: Mr. F. J. Hedden.

Recent advances in cerebral palsy: Dr. B. Epstein.

Repair of the hand in children after common domestic accidents: Mr. D. H. Walker.

Film: Rehabilitation in industry: Mr. M. Singer.

Life and contribution to orthopaedic surgery of Albert Hoffa—centenary 1859-1959: Prof. C. E. Lewer Allen.

OTORHINOLARYNGOLOGY

Monday 28 *September*

Transmeatal tympanotomy: Dr. M. Jackson.

Secretional anoxia and tracheotomy: Dr. D. V. Maytham.

Friday 2 *October*

The treatment of tetanus neonatorum: Dr. R. Wright.

Tuesday 29 *September*

Group discussions.

Wednesday 30 *September*

Group discussions.

PAEDIATRICS

Monday 28 *September*

Herpes simplex stomatitis in children: Dr. J. D. L. Hansen.

Disseminated herpes simplex infection: Dr. D. McKenzie.

Problems in the diagnosis and management of infantile gastro-enteritis: Dr. M. D. Bowie.

Tuesday 29 *September*

Ureterosigmoidostomy with perineal colostomy in vesical exstrophy: Mr. C. C. Wiggishoff.

An evaluation of the results of surgery in 1,000 cases of congenital hypertrophic pyloric stenosis: Dr. T. Meyer.

Ano-rectal malformations: Prof. J. H. Louw.

Wednesday 30 *September*

The feeding of premature babies in the neonatal period: Prof. F. J. Ford.

Iron-deficiency anaemia in Cape Coloured and African children: Dr. P. Lanzkowsky.

Coxsackie myocarditis: Drs. P. V. Suckling and L. Vogelpoel.

Friday 2 *October*

The treatment of tetanus neonatorum: Dr. R. Wright.

Recent advances in cerebral palsy: Dr. B. Epstein.

Repair of the hand in children after common domestic accidents: Mr. D. H. Walker.

Cardiac arrest—a review of 300 cases: Drs. O. V. S. Kok and C. Kitay.

Blood loss in paediatric surgery: Dr. J. A. Pretorius.

PATHOLOGY

Monday 28 *September*

Carcinoma *in situ* of the cervix: Prof. T. Antoine (Vienna).

A comparative study of the incidence, histological distribution and relation to cirrhosis of liver siderosis in autopsies of the 3 racial groups in Cape Town: Dr. C. J. Uys.

Wednesday 30 *September*

Transplantable suprarenal tumour in the rat: Dr. H. Stewart (USA).

PUBLIC HEALTH, INDUSTRIAL AND MILITARY MEDICINE

Monday 28 *September*

History of trachoma in South Africa: Dr. R. St. H. Warren.

Prevention and cure of trachoma: Drs. J. G. Scott and I. Taylor.

The result of the first field trial in treatment of trachoma: Dr. J. G. Scott.

Film: Trachoma in South Africa: Dr. D. Ferguson.

Health education in South Africa: Dr. D. Ferguson.

The organization of health services in the Union of Soviet Socialist Republics: Dr. H. H. Eiselen.

Activities of the National Cancer Association of South Africa: Dr. L. S. Robertson.

Wednesday 30 *September*

Space medicine: Major J. Horak.

Film: Frontier of the future (aero-medical and space medicine research): Major J. Horak.

Friday 2 *October*

Psychiatric approach to tuberculosis: Dr. B. W. Crowhurst Archer.

Film: Rehabilitation in industry: Mr. M. Singer.

RADIOLOGY

Monday 28 *September*

Lung cancer among White South Africans: Dr. G. Dean.

Tuesday 29 *September*

The leukaemias: Dr. M. M. Suzman.

Observations on the value of cephalo-pelvimetry undertaken during trial labour: Prof. E. D. Crichton.

Wednesday 30 *September*

Hypertension with unilateral kidney disease: Drs. A. J. Tinker and M. B. M. Denny.

Further observations in the radiological investigation of peripheral vascular disease: Dr. M. B. M. Denny.

Film: The technique of the aortogram and nephrogram: Dr. M. B. M. Denny.

Transplantable suprarenal tumour in the rat: Dr. H. Stewart (USA).

Friday 2 *October*

Mammary cancer: Prof. R. McWhirter (UK).

The place of intravenous cholecyst-cholangiography in biliary surgery: Dr. E. Price.

Radiotherapy of the common primary malignant conditions of the skin: Dr. M. Weinbren.

SURGERY

Monday 28 *September*

Treatment of sigmoid volvulus: Mr. J. Nayman.

Exploration of the common bile duct with special reference to a combined supra- and transduodenal approach: Mr. F. A. K. van Wyk.

A preliminary report on investigations into the pathogenesis of peptic ulcers: Prof. D. J. du Plessis.

The aetiology and management of gravitational ulcer: Mr. J. C. Allan.

Tuesday 29 *September*

Ureterosigmoidostomy with perineal colostomy in vesical exstrophy: Mr. C. C. Wiggishoff.

An evaluation of the results of surgery in 1,000 cases of congenital hypertrophic pyloric stenosis: Dr. T. Meyer.

Ano-rectal malformations: Prof. J. H. Louw.

Wednesday 30 *September*

Hypertension with unilateral kidney disease: Drs. A. J. Tinker and M. B. M. Denny.

Further observations in the radiological investigation of peripheral vascular disease: Dr. M. B. M. Denny.

Film: The technique of the aortogram and nephrogram: Dr. M. B. M. Denny.

Friday 2 *October*

The antigenic effects of implanted arterial heterografts: Mr. J. C. Allan.

Reconstructive arterial surgery: Mr. H. Gaylis.

Film: (Mr. H. Gaylis).

Some aspects of the surgical treatment of peptic ulcers: Prof. D. J. du Plessis.

A new local treatment for burns and scalds: Mr. H. F. Kamp.

Repair of the hand in children after common domestic accidents: Mr. D. H. Walker.

Mammary cancer: Prof. R. McWhirter (UK).

The place of intravenous cholecyst-cholangiography in biliary surgery: Dr. E. Price.

Cardiac arrest—a review of 300 cases: Drs. O. V. S. Kok and C. Kitay.

Film: Cardiac arrest: Drs. O. V. S. Kok and C. Kitay.

Film: Treatment of cardiac arrest: Sir Russell Brock (UK).

Blood loss in paediatric surgery: Dr. J. A. Pretorius.

Radiotherapy of the common primary malignant conditions of the skin: Dr. M. Weinbren.

THORACIC SURGERY

Monday 28 *September*

Atrial septal defect: Drs. M. M. Zion, J. L. Braudo and B. A. Bradlow.

Inter-atrial septal defects development: Mr. D. N. Fuller.

Open-heart surgery in the management of atrial septal defects: Mr. W. L. Phillips.

Open-heart surgery using the disc oxygenator and roller type pumps: Mr. L. A. du Plessis.

Film: Preparation and operation of the disc type of oxygenator: Mr. L. A. du Plessis.

Tuesday 29 *September*

The aortic valve, with special reference to the surgical correction of aortic insufficiency: Drs. R. de Villiers and C. N. Barnard.

Film: Drs. R. D. de Villiers and C. N. Barnard.

The surgical treatment of aortic stenosis: Sir Russell Brock (UK).

Artificial heart valves: Dr. B. Dreyer.

Films: Artificial heart valves cine-cardiography: Dr. B. Dreyer.

Wednesday 30 *September*

Surgical correction of pectus excavatum: Mr. W. L. Phillips.

Film: (Mr. W. L. Phillips).

Complete correction of some congenital heart lesions: Dr. C. N. Barnard.

Surgery of congenital aortic stenosis: Mr. D. I. Adler.

Film: Repair of an atrial septal defect using heart-lung machine: Mr. W. L. Phillips.

Friday 2 *October*

The surgical treatment of Fallot's tetralogy: Sir Russell Brock (UK).

The mitral valve, with special reference to problems in the surgical correction of mitral regurgitation: Drs. M. B. McKenzie and C. N. Barnard.

Film: Mitral valve study: Dr. M. B. McKenzie.

Film: Open-heart surgery with the use of the Gross type pump-oxygenator: Mr. D. N. Fuller.

Cardiac arrest—a review of 300 cases: Drs. O. V. S. Kok and C. Kitay (with film).

Film: Treatment of cardiac arrest: Sir Russell Brock (UK) (9 mins.)

Blood loss in paediatric surgery: Dr. J. A. Pretorius.

UROLOGY

Monday 28 *September*

A review of eight years' work (1951-1958) on cancer of the cervix uteri: Prof. J. T. Louw.

Drainage tubes in urology: Mr. J. D. Joubert.

Tuesday 29 *September*

Ureterosigmoidostomy with perineal colostomy in vesical exstrophy: Mr. C. C. Wiggishoff.

Wednesday 30 September

Urinary infection associated with catheterization in gynaecology and obstetrics: Dr. A. Viljoen.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES REMOVED FROM THE LIST

The name of the following society has been removed from the list of approved medical aid societies as from 1 July 1959:

L. H. Martinusen Medical Aid Society, P.O. Box 64, Denver, Transvaal.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
8 July 1959

MEDIESE HULPVERENIGINGS VAN DIE LYS GESKRAP

Met aanvang 1 Julie 1959 is die naam van die volgende hulpvereniging van die lys van goedgekeurde mediese hulpverenigings geskrap:

L. H. Martinusen Medical Aid Society, Posbus 64, Denver, Transvaal.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
8 Julie 1959

MEDIESE HULPSKEMAS GEDRYF DEUR VERSEKERINGSMAATSKAPPYE

'n Groot mate van verwarring het ontstaan oor die houding wat lede van die Mediese Vereniging behoort in te neem ten opsigte van mediese hulpskemas wat gedryf word deur versekeringsmaatskappye. Op sy laaste vergadering het die Federale Raad hierdie saak bespreek nadat die Sentrale Komitee in verband met Kontrakpraktyk 'n rapport oor hierdie saak opgestel het na aanleiding van navrae oor die optrede van sekere mediese versekeringskemas.

Die beleid van die Mediese Vereniging is om die vorming van mediese hulpverenigings aan te moedig en hierdie beleid is konsekwent gevolg deur die erkenning van tyd tot tyd van addisionele verenigings as hulle voldoen het aan die vereistes van die Vereniging. Hierdie vereistes het hoofsaaklik te doen met die inkomstegroep van die lede—wat die middel en laer inkomstegroep behoort te wees—en met die direkte en volle vereffening van die rekeninge van dokters as hulle opgestel word ooreenkomstig die Tarief vir Goedgekeurde Mediese Hulpverenigings.

Mediese hulpskemas wat deur versekeringsmaatskappye georganiseer is, het onlangs ontstaan. Hierdie skemas is oop vir alle inkomstegroepe. Sommige lede het die opvatting dat aangesien hierdie skemas nie genoegsame amptelike ‚erkenning' geniet nie, hulle niks met die skema te doen moet hê nie en nie eers lede wat daaraan behoort moet behandel nie.

Tot dusver het die versekeringskemas aan lede self die voordele betaal wat aan hulle toekom. Die voordele verteenwoordig 'n gedeelte van die dokter se rekening en die lid moet die verskil uit sy eie sak betaal. In een geval stuur die versekeringsmaatskappy die tjek aan die lid alhoewel dit uitgemaak is aan die persoon aan wie die betaling toekom sodat die dokter ten minste verseker is dat hy die grootste deel van sy gelde sal ontvang.

Die vraag het ontstaan of die Mediese Vereniging een of ander vorm van erkenning aan die mediese versekeringskemas behoort te gee. Daar is gevoel dat dit nie moontlik is om dit te doen nie aangesien die skemas voorsiening maak vir persone uit alle inkomstegroepe sonder enige kontrole, en daar kan geen sprake wees daarvan dat die voorkeurtarief toegepas word nie tensy 'n vereniging voldoen aan die reëls van die Mediese Vereniging. Aan die anderkant word dit erken dat die versekeringskemas

onder andere voorsiening maak vir persone wat nie by goedgekeurde verenigings kan aansluit nie, byvoorbeeld persone wat vir hulself werk, en die hulp wat dus aan lede gegee word om hulle mediese onkoste te betaal kan slegs in die voordeel van die professie wees aangesien die gelde gebaseer is op gelde wat deur private pasiënte betaal word.

Die Federale Raad, in die besef dat mediese versekeringskemas waarskynlik as permanente instellings gekom het, het die volgende besluit geneem:

> ‚Dat hierdie Federale Raad alle vorms van versekering teen siekte verwelkom en dat die Raad dus 'n spesiale Versekeringskomitee aanstel met die doel om permanente kontak met alle versekeringsmaatskappye te bewerkstellig ten opsigte van al hulle aktiwiteite. Die naam van hierdie Komitee sal wees die Versekeringskomitee van die Federale Raad.'

Lede van die Mediese Vereniging word dus aangeraai om seker te maak dat pasiënte wat hulself voorstel as lede van mediese hulpverenigings, wel lede is van verenigings wat amptelik goedgekeur is deur die Mediese Vereniging voordat aansoek gedoen word om toepassing van die voorkeurtarief. Lede van ander verenigings moet dus behandel word as gewone private pasiënte wat sodanige vergunning kan kry as wat die praktisyn self gewillig is om toe te staan in elke besondere geval.

Mediese Huis
Kaapstad
25 Junie 1959

L. M. Marchand
Medesekretaris

PASSING EVENTS : IN DIE VERBYGAAN

42nd Medical Congress (*M.A.S.A.*), *East London*, 27 *September—3 October* 1959. On Friday, 2 October 1959, at 8 p.m. a special social gathering will be held for members of Congress who hold a degree from one of the Scottish universities, or who studied at a Scottish university for the L.R.C.P. & S. of Glasgow or Edinburgh. This function is to be held in the Carlton Banquet Hall. Tickets £1 1s. 0d. each. Each member who attends may bring a guest. All doctors who are interested are requested to communicate with Dr. H. Dyke, c/o Medical Congress Office, 10-12 Oxford Street, East London.

* * *

Dr. Allan Nestadt, M.B., Ch.B., M.R.C.P. (Edin.), has recently commenced practice as a paediatrician at 31 Medical Centre, Field Street, Durban. Telephone numbers: Rooms 69085, residence 57844.

* * *

Dr. H. Ludwig Naudé, medical officer to Rhodesia Cement Limited since 1956, and previously in practice at Balfour, Transvaal, is relinquishing this position and will be entering partnership practice with Dr. William Schneeberger, in Chingola, Northern Rhodesia, as from 1 August 1959.

* * *

Dr. Harold O. Hofmeyr, of Cape Town, has recently returned to South Africa after a 2-months visit to the USA. Part of the time which Dr. Hofmeyr spent overseas was devoted to visiting some of the leading cardiac centres of the USA. Dr. Hofmeyr spent some time with Drs. Paul White and Ancel Keyes, and he visited the Mayo Clinic.

* * *

South African Society of Anaesthetists, Cape Western Sub-group. The next meeting of this Sub-group will be held on Monday 3 August in the small A-floor lecture theatre, Groote Schuur Hospital, Observatory, Cape, at 8.15 p.m. Dr. J. Abelsohn will speak on 'Unusual incidents and accidents in anaesthesia'. All who are interested are invited to attend this meeting.

* * *

Cape Western Branch (*M.A.S.A.*). The monthly meeting of this Branch will be held in the Physiology Lecture Theatre, Medical School, Observatory, Cape, on Friday 31 July 1959 at 8.15 p.m. Drs. E. M. Sandler and J. N. de Klerk and a member of the Cape Bar Council will be the speakers in a symposium on 'Artificial insemination donor'.

* * *

Dr. Herman Claassens, M.Med. (O. en G.), M.R.C.O.G. van Kaapstad, woon die Britse Kongres van Obstetrie en Ginekologie te Cardiff, en ook die Britse Mediese Kongres te Edinburgh by. Teen die einde van Julie hoop hy om na die Universiteit van Washington te vertrek as besoekende genoot. Hy hoop ook om 'n aantal klinieke in Boston en New York te besoek. Dr. Claassens is van plan on in Januarie 1960 in Kaapstad as spesialis te begin praktiseer te Mediese Sentrum.

* * *

Dr. Harding leRiche, B.Sc., M.D. (Rand), M.P.H. (Harvard), has been appointed to the post of Professor of Public Health, School of Hygiene, University of Toronto. He has also recently been nominated to serve on the Canadian Council on Nutrition to represent the Canadian Medical Association. For a number of years he has been research consultant to Physicians' Services Inc., a medically-sponsored insurance organization for prepaid medical services.

* * *

Dr. Norman Klass, B.A., M.B., B.Ch., D.Phys. Med., who has just completed a 3-months' study tour of university physical medicine departments in Israel, Turkey, Britain and the Continent, has now returned to Johannesburg.

Dr. Norman Klass, B.A., M.B., B.Ch., D.Phys. Med., wat onlangs 'n 3-maande-studiereis van fisiese geneeskunde aan universiteitsdepartemente in Israel, Turkye, Brittanje en die Vasteland voltooi het, is nou terug in Johannesburg.

* * *

Leprosy. The Director of the World Health Organization for Africa, Dr. F. J. C. Cambournac, has announced that out of the 10-12 million lepers in the world about 2,300,000 cases are to be found in Africa south of the Sahara and that about half of this number are already under treatment. The mass application of sulfone treatment has considerably extended the battle against leprosy in Africa and it is estimated that if operations continue at the present satisfactory rate nearly all the lepers on this continent will be under treatment within the next few years.

* * *

Dr. Jan A. van der Merwe, M.B., Ch.B., F.R.C.S. (Edin.) wat onlangs teruggekeer het van nagraadse studie aan die Royal National Ortopediese-Hospitaal, Londen, en 'n besoek aan ortopediese klinieke in die V.S.A., praktiseer nou as ortopediese chirurg in vennootskap met dr. T. B. McMurray te Mediese Sentrum 409, Heerengracht, Kaapstad. Telefoon: 2-6151/2.

Mr. Jan A. van der Merwe, M.B., Ch.B., F.R.C.S. (Edin.), who has recently returned from postgraduate study at the Royal National Orthodaedic Hospital, London, and a visit to orthopaedic clinics in the USA, has joined Mr. T. B. McMurray as an orthopaedic surgeon at 409 Medical Centre, Heerengracht, Cape Town. Telephone 2-6151/2.

PHARMACEUTICAL NEWS : FARMASEUTIESE NUUS

RETIREMENT : MR. CECIL ARGENT

After 50 years of service with the Company of Allen & Hanburys Ltd., Mr. Cecil Argent, General Manager, Surgical Division for Southern Africa, retired on 30 June 1959. Mr. Argent gained his early training in the Company's showrooms in Wigmore Street, London, where his daily contacts with many eminent surgeons laid the foundation of his unique understanding of the surgeon's requirements. He played a major part in developing Allen & Hanburys surgical activities in Johannesburg and Durban since he came to South Africa in 1913.

Mr. Argent is succeeded as Surgical General Manager by Mr. H. R. King, who is stationed in Johannesburg. Mr. D. McGennis takes charge of the surgical branch in Durban.

BOOK REVIEWS : BOEKBESPREKINGS

OPHTHALMOLOGY

Systemic Ophthalmology. 2nd edition. Edited by Arnold Sorsby. Pp. xiv.+682+(19). 277 figures. 118s. 3d. + 2s. 3d. postage. London: Butterworth & Co. (Publishers) Ltd. South African office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1958.

The first edition of Systemic Ophthalmology of 1951 immediately established itself as a reliable, lucid exposition of the wide field embraced by the study of ophthalmology today. The second edition will certainly prove equally popular. Under the same editorship, the whole subject has been brought completely up to date, with its ramifications into all branches of medical knowledge. Most chapters appear under the original authorships, but several chapters have been entirely rewritten in the light of modern knowledge by new and equally distinguished contributors, and with fewer pages withal. The work is highly recommended as a reference book adequately surveying the interrelationship of ocular conditions and the rest of the body.

L.S.

OPERATIEWE VERLOSKUNDE

Die Geburtshilflichen Operationen. Ihre Ausführung und Anwfendung. Ein Lehrbuch für Studenten und Gebrauchsbuch ür-Ärzte. Von Prof. Dr. H. Martius. 8., Verkürzte und verbesserte Auflage. xvi+281 Seiten. 253 Abbildungen. DM 29.60. Stuttgart: Georg Thieme Verlag. 1958.

Hierdie is natuurlik een van die klassieke boeke oor die onderwerp van operatiewe verloskunde. Die opstelling, feite-materiaal en afbeeldings was vanaf die eerste uitgawes van so 'n aard dat daar oor die laaste aantal jare feitlik geen verandering in die boek nodig was nie. Dit is een van die seldsame boeke wat sterk aanbeveel kan word by die mediese student en terselfdertyd van groot waarde is vir die nagraadse student en vir die spesialis-verloskundige. Dit is 'n boek wat maklik gelees, met helderheid bestudeer en met 'n mate van gerief onthou kan word.

Die eerste een-derde van die boek word gewy aan die anatomie en fisiologie van die baring. Daarop volg die verloskundige metodiek, en ingrepe en operasies by die abnormale verloskunde.

Die feitemateriaal wat in die teks bespreek word is uit die aard van die saak nie juis verskillend van die wat in die Engelse en Amerikaanse teksboeke behandel word nie, maar die boek van Martius verskil veral in twee opsigte:

Eerstens is dit 'n boek wat geskryf is om te bestudeer, en die materiaal word so aangebied dat die studie van die onderwerp aansienlik vergemaklik word. Hierby word ruim gebruik gemaak van verskillende soorte druktipes, van indeling van paragrawe en van uitstekende afbeeldings wat die teks verhelder en aanvul. Die manier waarop die belangrike feite op elke bladsy uitstaan verdien nabootsing in ander teksboeke.

Tweedens word die praktiese ingrypende verloskunde behandel volgens die opvattings wat heers op die Vasteland van Europa. Waar dit in sekere opsigte verskil van die verloskunde soos gedoseer in Engeland, is dit goed dat ons ook dit wat goed is in die Europese benadering, indikasie-stelsel, en metodiek onder die oë gebring kry.

Martius is die outeur van meer as een boek wat die ginekologie en die verloskunde behandel, maar 'Geburtschilflichen Operationen' is sekerlik sy beste.

D.A.H.duT.

CHIRURGIESE ONDERSOEKMETODES

Chirurgiese Ondersoekmetodes. Deur Charles F. M. Saint, *C.B.E.*, M.D., M.S., F.R.C.S. (Eng.), F.R.A.C.S. (Hon.), en Jan H. Louw, Ch.M. Pp. xiv+149. 21s. Kaapstad, Wynberg, Johannesburg: Juta en Kie. Beperk. 1959.

Die bekende handleiding vir studente (Surgical Note-taking) deur prof. C. F. M. Saint, voormalige professor van chirurgie aan die Universiteit van Kaapstad en prof. J. H. Louw, die huidige bekleër van hierdie leerstoel, het nou ook in Afrikaans verskyn (Chirurgiese Ondersoekmetodes).

Daar het baie geslagte van studente deur die Universiteit van Kaapstad gegaan wat van hierdie nuttige boekie veel voordeel en plesier gehad het. ‚Chirurgiese Ondersoekmetodes' is, soos die titel aandui, nie 'n uitgewerkte stelsel van die teorie en die praktyk van die chirurgie nie, maar dit is bedoel om die student te help om die kuns en die tegniek van die diagnose van 'n siektetoestand te bemeester deur sistematies te werk te gaan by die afneem en opskryf van notas in die siekesaal. As sodanig is dit 'n waardevolle boek en het dit reeds al sy waarde in die verlede bewys.

Van spesiale belang vir ons is dat hierdie boek nou ook in Afrikaans verskyn het en daardeur 'n definitiewe bydrae maak tot die nog skrale oes van mediese wetenskaplike boeke in Afrikaans. Daar sal baie wees wat teen die Afrikaans wat gebruik word besware sal kan opper—dit word verwelkom. Dat ons nou die stadium bereik het waarin 'n mens haas niks in Afrikaans kan skryf op mediese gebied sonder dat daar 'n sterk verskil van mening ontstaan nie, is 'n bewys dat dit goed gaan met die mediese terminologie in Afrikaans. Daar is met ander woorde 'n lewendige belangstelling in hierdie saak.

Ons wil die gebruik van ‚Chirurgiese Ondersoekmetodes' sterk aanbeveel by alle studente van die vak en by almal wat te doen het met die onderrig van die chirurgie.

A.P.B.

ORTHOPAEDICS

Outline of Orthopaedics. 2nd edition. By John Crawford Adams, M.D., F.R.C.S. Pp. vii+428. 301 figures. 35s. net + 1s. 11d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

That the 'Outline of Orthopaedics' has appeared in a new edition barely 2 years after its first publication is high tribute to the excellence of this small but comprehensive volume which Mr. Crawford Adams has written. All aspects of orthopaedic surgery, apart from trauma, are completely covered and succinctly put down and the concepts of pathogenesis and treatment are up to date. One is able to obtain a clear picture of each disorder from aetiology to prognosis and treatment within a few minutes, without having to wade through pages of irrelevant detail. This makes it invaluable to the overburdened medical student and the busy general practitioner. The disorders of the locomotor system are first discussed in general terms. This is followed by regional accounts, each preceded by a description of the method of examination of the area, together with a table listing the disorders to be considered. The book has been completely revised and advances during the past few years are indicated where relevant.

To those uninitiated in orthopaedics this is an excellent volume, which will give a balanced approach and place the subject in its proper relationship to the other fields of medicine. The volume has been excellently printed on fine art paper and is profusely illustrated with excellent diagrams, line drawings, photographs and X-rays. These make for a clearer understanding of each disorder. The diagrams and drawings, in particular, enhance the value of this work.

I.J.

BACTERIA IN RELATION TO NURSING

Duke's Bacteria in Relation to Nursing. 3rd edition revised by Stanley Marshall, M.D., B.S. (Lond.), M.R.C.S. Pp. viii+216. 18 illustrations including 12 in colour. 21s. net. London: H. K. Lewis & Co. Ltd. 1958.

This book should be very useful to all nurses interested in this aspect of their profession, especially to sister tutors. The new edition includes additions covering recent discoveries with special reference to viruses and antibiotics. The rest of the book has not been materially altered, but certain minor alterations might bring it more up to date, e.g., the use of the now universally used 'doubling dilution' method of agglutination test instead of the old type of Widal reaction, as an example of a serological test. The description of an autoclave in a paragraph headed 'intermittent sterilization' is misleading. The substitution of the term 'megalosporon' for the 'trichophyton' of the old edition is no improvement. But these are minor faults in an otherwise useful book.

P.D.

HAVING A BABY

Having a Baby. 2nd edition. By J. F. Robinson, M.B., Ch.B. Pp. viii+100. 23 figures. 6s. 6d. net + 10d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

This little book may be recommended confidently to all young married couples who wish to start a family. The opening chapters

describe both simply and adequately what they need to know about conception and contraception. Subsequently, and indeed herein lies the main purpose of the book, the author describes for the benefit of the expectant mother the progress of normal pregnancy, the growth of the foetus, and finally labour and the birth of the child.

All the many aspects of having a baby which occupy the young mother-to-be are dealt with. There is a wealth of information concisely and pleasantly set down, while the advice given is at all times most useful and sensible. The price is reasonable.

E.M.S.

HANDBUCH DER TUBERKULOSE

Handbuch der Tuberkulose. Band I. Allgemeine Grundlagen. Herausgegeben von Prof. Dr. J. Hein, Prof. Dr. Dr. h.c. H. Kleinschmidt Prof. Dr. E. Uehlinger. xvi+832 Seiten. 244 Abbildungen. Ganzleinen DM 178. Subskriptionspreis DM 142.40. Stuttgart: Georg Thieme Verlag. 1958.

It is an impossible task in a brief review to render justice to such a tremendous volume on so many different aspects of tuberculosis. The story dates from 2500 B.C. and depicts the devastating terminal effects as revealed by Egyptian mummies, especially of the XXI Dynasty and Malum Potti for instance. In subsequent chapters the authors refer to the great pioneers of the past, paying tribute to their valuable contributions. Methods of examination, morphology, diagnosis and prognosis are reviewed and the significance of prevention emphasized.

The reviewer must be content to say that this volume is a perfect combination of history, theory, diagnosis and treatment—one which cannot be summarized in a few words, even to indicate to the interested reader how much he will miss unless he studies the book for himself.

J.H.

DUKE-ELDER'S NEW SYSTEM OF OPHTHALMOLOGY

System of Ophthalmology. Edited by Sir Stewart Duke-Elder, *G.C.V.O.*, M.A., LL.D., Ph.D., D.Sc., M.D., D.M., F.R.C.S., F.R.C.S.E., F.A.C.S., F.R.A.C.S. *Vol. I. The Eye in Evolution.* Pp. xvi+843. 902 figures. 15 Coloured plates and 350 Marginal Illustrations. 126s. net. London: Henry Kimpton. 1958.

Since its first appearance, Duke-Elder's 'Text-book of Ophthalmology' in seven volumes, has become the standard reference book on ophthalmology in the English language. As much of the content has become out of date in such a rapidly expanding subject as ophthalmology, the whole work has been rewritten and expanded by Duke-Elder and his colleagues of the Institute of Ophthalmology into a library of 15 volumes. The present volume is the first of the series. Written by the master himself, in his inimitable style, this masterpiece of the comparative anatomy and physiology of the eye is an expansion of the first 22 pages of volume I of the Text-book. It is profusely illustrated and there are striking marginal illustrations of common and strange-looking birds and animals and fishes. The practising ophthalmologist may not find this volume of such great clinical use but it certainly abounds in interesting and informative material and is a promise of more exciting things to come. It is certain that as they appear, each volume of the System will find its way onto the shelf and into the mind and affection of every ophthalmologist.

L.S.

X-RAY DIAGNOSIS

A Text-book of X-ray Diagnosis. By British authors in 4 volumes. 3rd edition. Edited by S. Cochrane Shanks, *C.B.E.*, M.D., F.R.C.P., F.F.R. and Peter Kerley, *C.V.O.*, *C.B.E.*, M.D., F.R.C.P., F.F.R., D.M.R.E. *Vol.* 3. *The Abdomen.* Pp. xvi+883. 802 illustrations. £6 0s. 0d. net. London: H. K. Lewis & Co. Ltd. 1958.

Volume 3 of this excellent, well established and well tried text-book, is now available. Volumes 2 and 4 are still in preparation. Those familiar with the earlier editions, particularly the second, will appreciate that the mixture is essentially as before. The book generally is larger but large slices of it have been carried forward; this is also true of the illustrative material.

It seems a pity, however, that a work of this standing still fails, even on its 3rd edition, to introduce uniformity into the reproduction of radiographs. To find positive and negative reproductions of X-rays side by side on the same page (p. 545), is particularly irritating—at any rate to the reviewer, who likes barium and bones in white and wind in black. In a book of this calibre certain other small errors should have been eliminated in the proof stages; e.g. the reproducing of serial studies upside down of a gall-bladder designed to show floating stones (p. 494), and spelling mistakes such as pleurality for plurality (p. 558).

Apart from these quibbles, which are probably of no great importance, there is little with which to find fault. There can be no doubt that this volume should be included on the shelves of radiology students and of all practising radiologists.

W.J.L.

YEAR BOOK OF EAR, NOSE AND THROAT AND MAXILLOFACIAL SURGERY, 1958

The Year Book of the Ear, Nose and Throat and Maxillofacial Surgery, 1957-58. *The Ear, Nose and Throat.* Edited by John R. Lindsay, M.D. *Maxillofacial Surgery.* Edited by Dean M. Lierle, M.D. and William C. Huffman, M.D. Pp. 383. 96 figures. $7.50. Chicago: The Year Book Publishers, Inc. 1958.

The appearance of this and forthcoming volumes, dissociated from ophthalmology, is to be applauded; so is the decision to incorporate maxillo-facial surgery. However, maxillo-facial surgery accounts for only a small portion of the contents. In this part of the book, malignant disease of the nose and paranasal sinuses, the oral cavity including the tongue, and the larynx, pharynx and oesophagus, are adequately covered. In this section, too, benign diseases of the larynx are discussed and tumours of the salivary glands receive full attention. None of these subjects can be included in the province of the maxillo-facial surgeon and should be part of the ear, nose and throat section. Reconstructive surgery of protruding ears and rhinoplasty is included and, of course, fractures of the upper and lower jaws; also derangements of the temporo-mandibular joints, surgery of cleft palate and, lastly, surgical treatment of acne.

In the ear, nose and throat section, the latest advances in aural surgery, such as stapes mobilization and tympanoplasty, are described, but the material covered is out-of-date and of little help to the aural surgeon. On the whole this volume is disappointing.

B.T.B.

UROLOGY YEAR BOOK 1959

The Year Book of Urology 1958-59. Edited by William W. Scott, M.D., Ph.D. Pp. 364. 83 figures. $7.50. Chicago: The Year Book Publishers, Inc. 1959.

In maintaining its high standard as a comprehensive review of World urological literature, the publishers, and in particular Dr. W. W. Scott as editor, are once more to be congratulated. This is a Year Book which I look forward to each year and for which, in fact, I have a standing order with a local medical bookseller.

The Year Book as usual covers genito-urinary diseases in the wide sense, in that it encompasses teaching, research and experimental surgery, and allied medical and endocrinological subjects, as well as all the usual problems of the practising urologist. Its wide scope will enhance its reputation as a most useful reference for many general practitioners, surgeons, physicians and endocrinologists.

I believe I voice a genuine difficulty when I mention that one has considerable misgivings when trying to assess the true value of what one reads in medical journals. One is tempted to discard the value of an article unless one knows the author or the institution from whence it emanates. This is where the Year Book of Urology is so extremely helpful. One is conscious throughout of the watchful eye and the kindly, yet critical supervision of the editor, whose footnotes put statements and claims in their correct perspective.

P.J.M.R.

YEAR BOOK OF DRUG THERAPY 1959

The Year Book of Drug Therapy 1958-59. Edited by Harry Beckman, M.D. Pp. 569. 43 figures. $7.50. Chicago: The Year Book Publishers, Inc. 1959.

Those who are familiar with the Year Book series will not be disappointed in this volume dealing with drug therapy. As is to

be expected in a subject that is rapidly expanding, and advancing on many fronts, there is much that is new. The reader is provided with good abstracts from a great variety of journals, most of which can only be seen by those who 'live' in well-stocked medical libraries. In the present volume there are 493 articles from 23 countries and 108 journals. The field is considered up to September 1958. Additional useful items are the subject and author indexes, 43 figures, and numerous tables.

A valuable feature is the editorial comments (all too few) which indicate, for example, that a particular drug is really of value, possibly better than its predecessors, or that a particular study is unacceptable because the investigation was uncontrolled. Criticism is particularly levelled against the spate of publications covering hurried trials of so-called 'tranquillizers' and 'energizers'; 'there quickly follows a breathless report transparently betraying its author's desire to get into print before the other fellow does'. The Year Book is not merely a collection of abstracts.

N.S.

CARDIOVASCULAR REFLEXOGENIC AREAS

Reflexogenic Areas of the Cardiovascular System. By C. Haymans, M.D. and E. Neil, M.D., D.Sc. Pp. viii+271. 89 figures. 56s. net. London: J. & A. Churchill Ltd. 1958.

This book is written by a professor of pharmacology and a professor of physiology and it is difficult reading. To the experimental scientist, it will be of inestimable value, but the clinician will find the subject matter confusing. The literature is extensively reviewed, even when contradictory, and often no clear statement of the authors' own views is forthcoming. In the reviewer's opinion, a short summary at the end of each chapter, of the most important views expressed would add greatly to the value of the book. The names quoted are at times most irritating and detract from the interest of the text. The clinical interest of the book is limited, for most of the text is taken up on animal work. The sections on the 'carotid sinus syndrome' and 'Takayashu's disease' are very incomplete, but this is perhaps justifiable in a book devoted to physiology and pharmacology. 'Neurogenic hypertension' is well done, but as the condition is merely of theoretical interest in human hypertension, this section again has a limited value.

The book is well produced on art paper and the illustrations are, for the most part, excellent. Despite its drawbacks, it serves as an excellent reference book and it is presumed that the extensive review of the literature was intended by the authors for this purpose. It should be readily available in all physiological laboratories where work on the baro- and chemo-receptors of the cardiovascular system is in progress, and should also be available to clinicians interested in the study of the cardiovascular systems and its reflexes.

B.A.B.

CHILD HEALTH

Child Health and Paediatrics. For nurses, health visitors and social workers. By R. McL. Todd, M.A., M.D., M.R.C.P., D.C.H. Pp. ix+238. Illustrations. 21s. net. London: William Heinemann Medical Books Ltd. 1958.

The proper care and nursing of children, particularly when they are ill in hospital, requires not only a love of children, but a great deal of specialized knowledge. This little book, written especially for nurses, health visitors and social workers, has much to recommend it. The material is up to date and well described, and care has been taken to cover essential problems. The chapter on the handicapped child is particularly well presented. This book should be very useful to those for whom it has been intended. Medical students, too, may find it a good introduction to their paediatric course.

I.M.

CLEFT PALATE AND SPEECH

Cleft Palate and Speech. 4th edition. By Muriel E. Morley, M.Sc., F.C.S.T. Pp. xx+271. 86 figures. 27s. 6d. net + 1s. 5d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

The fourth edition of this book, the most comprehensive and best known in its field, is welcomed. It is of particular interest to all those in medical fields and related ancillary services who are concerned in the treatment of these handicapped patients.

During the past decade, the many advances in cleft-palate surgery and modifications and changes in treatment have necessitated a new approach by the logopaedician. The goal has changed from intelligible speech to normal speech. Consequently, this book is of primary interest to the speech therapist, but the excellent sections on the incidence, aetiology and inheritance of cleft lip and palate are of wide interest to pathologists, paediatricians and all practitioners working with infants.

The section on anatomy and physiology of the nasopharyngeal closure mechanism is the finest and most comprehensive account of this region in the English language. A good understanding of this mechanism is a necessary basis for the understanding of many speech aberrations. The dental surgeon will find the account of obturators and the new light plastics of interest. The clear account of surgical techniques and post-operative assessment and the critical anaylsis of post-operative results are of major importance to surgeons working on cleft lip and palate.

The book is well published and clearly illustrated. The emphasis throughout is on practical management and team work. This new edition gives us the benefit of the author's vast experience at Newcastle-on-Tyne, and her concise, lucid account of every aspect of the cleft palate, make this book a 'must' for all those responsible for any phase of the treatment of these patients.

B.G.

CORRESPONDENCE : BRIEWERUBRIEK

MEDICAL GOLFING SOCIETY

To the Editor: There is a flourishing Medical Golfing Society in this Colony for the promotion of golf among medical and dental practitioners.

I wonder whether a society of like nature exists in South Africa with which we might reciprocate and whose members we could look forward to meeting when on leave in South Africa, or when they visit Kenya.

Charles J. Coghlan
Hon. Secretary,
Kenya Medical Golfing Society
P.O. Box 30024
Nairobi, Kenya
5 June 1959

[Members who are interested n this subject are requested to communicate with Dr. Coghlan.—*Editor*]

MENIÈRE'S DISEASE

To the Editor: In reference to a reader's question concerning treatment for a patient suffering from Menière's disease in the issue of the *Journal* of 20 June,[1] permit me to add my views to your reply.

In the first instance the diagnosis of unilateral Menière's disease should be established with a typical history, then by caloric tests, audiogram and recruitment assessment. Secondly, if there is little or no useful hearing, and the condition has failed to respond to a sedative with very large doses of nicotinic acid, Mygind's salt-free diet, fluid restriction plus ammonium chloride in large doses to displace the sodium ions which retain fluid, then, if the patient is sufficiently disabled, surgery is the treatment of choice.

A safe, easy and effective operation is extirpation of the endolymphatic ducts *via* the lateral semicircular canal through a simple cortical mastoid approach. The use of alcohol or cautery is regarded as too risky.

Cawthorne claims that 80% of cases respond to conservative treatment. Ancovert (BDH) should be tried. Histamine injections have also been used with a measure of success.

Mental assurance of the patient is most important in conservative treatment.

J. Fine

706 Medical Centre
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Johannesburg
27 June 1959

1. Questions Answered (1959): S. Afr. Med. J., 33, 524.